NORTH CAROLINA MEDICAL JOURNAL

OWNED AND PUBLISHED BY

THE MEDICAL SOCIETY OF THE STATE OF NORTH CAROLINA

VOLUME 9 MAY, 1948 NUMBER 5

PSYCHIATRY IN THE MODERN MEDICAL SCHOOL

ARTHUR H. RUGGLES, M.D.*

PROVIDENCE, RHODE ISLAND

Perhaps the title of this talk should be changed to "*Modern* Psychiatry in the Modern Medical School." The old descriptive psychiatry which is still taught in a few medical schools is not modern psychiatry, and should not be perpetuated in the modern school trying to train medical students in applied psychiatry. For too many years medical students were burdened with psychiatric terminology of a descriptive type and with classification of nervous and mental diseases on the basis of symptomatology. Now we know that the medical student must be interested in a dynamic concept which helps him to understand not so much whether an illness is a manic-depressive psychosis, a reactive depression, or an involutional melancholia, but rather why Mr. A or Mrs. B becomes depressed, apparently concerning a situation that is common to thousands of Mr. A's and Mrs. B's who do not break down. What is the physiologic or psychologic weakness that makes this individual vulnerable to depressive reactions?

Emphasis on Etiologic Diagnosis and Treatment

The modern medical school now knows that some mental illnesses are *directly* due to physical disorders, and teaches these facts. It teaches also how to remedy the underlying physical disorder through medical treatment, surgical interference, or dietary readjustment. There are many other nervous and mental illnesses, however, for which we do not know the exact etiology. In such cases we must constantly search for underlying factors—biochemical or psychologic, and sometimes both. The medical student must be given an understanding of the desire to escape from an unpleasant situation through the route of neurosis or psychosis, and must be taught how factors can be manipulated in order to get the patient to employ a more socially and medically acceptable method of meeting difficult situations.

Mr. S., a mild-mannered, industrious little man of 59 years, makes an extremely violent and aggressive suicidal attempt. Why should this Mr. Milktoast choose such an obviously aggressive action? The answer can not, in our modern teaching, be given by saying that he has a depression. It must go extensively into the background and source of his aggression, which proved to be directed against an extremely dominating and sadistic member of his family, for whom he had a terrific, but for many years controlled, hatred. This situation was brought out during a sodium amytal interview, when his inhibitory mechanisms were temporarily removed by the drug. Then we gained a scientific understanding of the case that made our treatment intelligent and effective.

Let us take another case, which illustrates the need for understanding, not only of the personality make-up, but also of the age periods in which certain types of mental illness are most common, and of etiologic factors, both physical and psychic.

Mrs. M., a 48-year-old, childless widow, has spent the last ten years, since her husband's death, employed in a most important

Read at the dedication exercises held at the opening of Graylyn, rehabilitation center of the Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, September 19, 1947.

* Superintendent of Butler Hospital, Providence, Rhode Island.

secretarial position. She has been outgoing, tending at times to show some overactivity and some overoptimism, but doing her work well. Her office associates notice that she is becoming a bit careless in both her work habits and her social relationships, as well as in her dress. They advise her to see her family doctor, saying that she is "on the verge of a nervous breakdown." A history is taken, a cursory physical examination is made, and she is told that she is working too hard and must rest for a while. During the prescribed two weeks' rest period, she is more and more active, spending money far too freely, and becoming increasingly disordered in her social engagements and in her dress. She begins to develop grandiose ideas, and telephones at all times of day and night to even casual acquaintances. In her written productions, her closest friends notice a marked disorder in her handwriting, as well as in what she attempts to say. She returns to her family doctor for a check-up, and he, appreciating the abnormal state of mind, sends her to a psychiatrist.

In this case the doctor must utilize to the full all known methods of diagnosis, in order to apply the appropriate treatment. Psychologically, the overactivity, some flight of ideas, and a grandiose trend of thinking and acting are apparent. Is this a nervous breakdown, or is it the result of underlying physical illness? A careful physical examination shows abnormal deep reflexes, irregular and abnormally reacting pupils, and occasional slurring in speech. These findings are all too often attributed to nervous exhaustion, but to the well-trained physician, who has been thoroughly grounded in medical school in the close relationship of physical disorders and mental reactions, they indicate the need for a laboratory examination of the blood and spinal fluid. This examination makes evident the diagnosis of general paresis, and the appropriate treatment for this disease may then be administered. Before we knew that this particular mental disorder was due to syphilis of the nervous system, the treatment was unavailing and the patient died a lingering and pathetic death in about two years. Today, with our knowledge of etiology and modern methods of treatment, the result in the majority of cases is very favorable.

This case illustrates the relationship between bodily disease and mental symptoms, and teaches the student that a good history and an understanding of the patient's psychologic make-up are necessary to explain the overactive, grandiose mental picture which developed when the normal inhibitory mechanisms were relaxed by disease. With such a case for teaching material, the instructor can discuss the whole subject of somatic and psychologic inter-relationships, and show the student how an accurate diagnosis is essential to adequate and effective treatment. The necessity for a thorough knowledge of nervous pathways, the method of attack and development of invading organisms, and the devastating results upon personality structure can be clearly illustrated.

Emphasis on the "Total Personality"

We have heard a great deal through the past years about the necessity of imparting to the medical student an understanding of the "total personality." Certainly the best teacher of psychiatry must make his pupils realize the need for obtaining psychologic, biologic and sociologic facts, and teach them how to put that factual material together to give a picture that makes of the patient a human being. The psychologic facts must include all his background of success or frustration, repression or regression, evasion or the attempt to face reality, as well as his insight. On the biologic side, the disfiguring facial birthmark, the handicap of diabetes, the deformity of infantile paralysis must be related to certain problems of behavior and to the patient's attempts at adjustment. The sociologic history includes the side of the railroad tracks on which the patient lives, the kind of group he is thrown with, the influence of poverty and bad housing. All these factors affect personality structure and must be given proper consideration in determining the causes of abnormal behavior.

Emphasis on "Psychosomatic Medicine"

We hear much of a recently coined term, "psychosomatic medicine." The psychiatrist has long understood that an injury to the knee may be related to behavior disturbances in certain individuals, and that mild or severe heart disease may sometimes produce emotional difficulties. We know too that prolonged emotional disturbances often create somatic disorders—for example, peptic

ulcers. As Dr. Edward A. Strecker has recently said,

"The real beginning of psychosomatics must be made in the preclinical years of medical education. Medicine not only has its physics or somatics but also its 'psychics' or psychogenetics. There could be a true and helpful teaching parallel. In anatomy there are livers, hearts and spleens. So too is there an anatomy of psychology to be studied, consisting of normal emotions, consciousness, memory and the like. There is a physiology which teaches how organs work. So too should there be taught a physiology of how mental functions work. Paralleling histology, the microscopic study of normal organs and tissues, the student should be made familiar with the finer divisions of mental functioning; for instance, remote and recent memory, the gradations of normal consciousness, the variety of emotional shadings and their somatic profiles. When the Department of Pathology is demonstrating gross morbid lesions, syphilitic aortitis, liver abscess and what not, the Department of Psychiatry should be demonstrating the gross pathology of the mind, pronounced emotional deviations such as profound melancholia or rage and fear with their component bodily expressions. Likewise should there be dual teaching of microscopic pathology, on the one hand, let us say, the study of a cross section of an arteriosclerotic vessel or the walls of an abscess, on the other a span of amnesia or the psychosomatics of anxiety due to repressed emotional conflicts.

"If the loaf of medical practice is to be thoroughly leavened, the medical student must be given from the very first week in medical school the opportunity of studying all of man and not only a hypothetical somatic half."[1]

Relation of Psychiatry to Other Fields

The modern medical school must see to it that there is a close relationship between the department of psychiatry and all the other departments. The psychiatric consultations requested by the medical and gynecologic services provide a fruitful opportunity for the training of the senior medical student. The sharing of experiences in both acute and chronic diseases between the medical men and the psychiatrist will produce great advances in the whole psychosomatic field and make for better medicine and better psychiatry. When I speak of the field of medicine, I am including all the medical and surgical specialties as well. The medical student should have instruction in the normal growth and development of children, as well as an understanding of some of the emotional problems of childhood. Thus a close liaison between the department of pediatrics and the department of psychiatry is necessary for the best teaching of both the future pediatrician and the future psychiatrist.

Fundamental training in neuro-anatomy, neurophysiology, and neuropathology is essential, for an understanding of the nervous pathways and their disorders is necessary to good clinical psychiatry. Just as many of our best internists have developed their diagnostic and therapeutic skills from a knowledge of human pathology, so, I believe, many of our best clinical psychiatrists must proceed to their positions of leadership in the profession by a very thorough understanding of neuro-anatomy and neuropathology.

While the best medical schools do not intend to make psychologists out of their graduates, it is nevertheless extremely important that the students be taught how to work with the psychologist, what important information the psychologist can contribute toward the understanding of human behavior, and how the psychiatrist can help the psychologist in making an accurate interpretation of the results of psychologic tests. From the psychology department, the medical student can gain much valuable instruction in the method of setting up scientific experiments, their control, and their evaluation. For the psychiatrist wishing to do research, this instruction is of the greatest fundamental value.

In teaching psychiatry today, we must give the student far more understanding of social factors and must teach him how to work with the psychiatric social worker. The student must also have some conception of the purpose and value of occupational therapy. A knowledge of the temperament of his patient will enable the doctor to recommend occupational therapy which will fit into the patient's needs and aid his recovery, rather than just occupy his time.

We must teach the student something of cultural anthropology, the differences in race cultures, and the problems of the minority groups. The medical student also needs some indoctrination in the psychiatric problems of industry. The maladjusted industrial worker plays an important part in labor turnover and labor unrest, and industry is calling for well qualified psychiatrists to help in their personnel problems.

Practical Clinical Experience

It almost goes without saying that medical students should have the most careful training in conducting psychiatric interviews, recording factual data, and interpret-

1. Strecker, E. A.: Psychosomatics; The Liaison between Internal Medicine and Psychiatry, J.A.M.A. 134:1520-21 (Aug. 30) 1947.

ing such data. This training can best be obtained either at the bedside of the patient or in the outpatient department, where the student can watch the expert psychiatrist conduct an interview, and can be taught, step by step, how the interpretation is arrived at. Since a large percentage of the patients the psychiatrist is going to see, especially in private practice, will be ambulatory patients, the experience gained in a well conducted neuropsychiatric outpatient department is invaluable.

Conclusion

I need not say to you that the program I have outlined is a big one, and that there are hardly enough hours in the undergraduate medical curriculum to provide adequate time for all this training. Psychiatric indoctrination should start in anatomy and physiology, however, and should go right on through all the courses, special emphasis being placed on clinical experience in the third and fourth years. It is the function of the medical school to train doctors to meet the needs of the community. If the medical school is to fulfill this mission in the highest degree, it must graduate medical students prepared to go on with their postgraduate training in psychiatry and ultimately to meet the great demand that exists today in the field of nervous and mental illnesses. In the past few years many of our schools have modernized their teaching, so that they are making a very great contribution in this direction, and I am sure that the Bowman Gray School of Medicine will take its place as a leader in training medical men to meet these increasing psychiatric needs of our state and nation.

The collection of information relative to a patient's complaint is an art which can be acquired only by constant and serious practice. Much patience and tact, an understanding and sympathetic attitude, and a good deal of time may be necessary in order to get the true facts and place them in proper perspective. Ability of this kind and a practical knowledge of the theory of evidence are absolutely essential for a successful barrister, and hardly less so for the sound practice of medicine.

Much important information can be obtained from an accurately taken history which can be gained in no other way. The early and even the more advanced phases of some diseases may express themselves only in symptoms, and the main or even the only diagnostic evidence reside in the history, for example, in some cases of angina of effort.—Leslie Hurley: The General Practitioner and the Specialist, M. J. Australia 1:67 (Jan. 17) 1948.

PSYCHIATRY COMES OF AGE

C. C. BURLINGAME, M.D.

HARTFORD, CONNECTICUT

Four decades ago, when I entered the field of psychiatry, it was the Cinderella of medicine. Rejected or ignored to a greater or lesser degree by the general public, it was even repudiated by the rest of medicine.

As a matter of fact, I believe I was the only medical student at that time who expressed a definite decision to become a psychiatrist — a departure which the hoary heads of tradition-bound medicine sternly looked upon as becoming an "alienist." I recall that mental institutions were often referred to as *psychopathic hospitals*—and, with all due respect to those who were struggling even then to improve conditions, I must confess that the institutions really were, for the most part, *very psychopathic!*

Since then, however, Cinderella has been admitted to society. During my professional lifetime as a psychiatrist, I have seen psychiatry become the "specialty-of-the-day."

I could not be more serious, more in earnest, nor more emphatic than when I say that I have been increasingly alarmed at the lack of discrimination with which newspapers, magazines, books, speakers, movies, and plays bandy psychiatric terminology, pseudo-psychiatric information, and, occasionally, a few psychiatric facts. Psychiatric literature, and public interest in it, is reaching the saturation point.

The Dangers of Popularity

Much as I would like to discuss why and how this sudden popularity came to psychiatry, I will abstain, and will deal only with the concomitant danger to psychiatry's future as a branch of medicine.

After nearly a century of obscurity, psychiatrists have come into their own. In some quarters, their services apparently have been recognized as indispensable. Even the least optimistic admit a growing lay understanding that there is no mystery about the so-called "mental illnesses," and that the prospect for recovery from several forms of mental illness compares favorably with the prospect of recovery from somatic illnesses.

Read at the dedication exercises held at the opening of Gravlyn, rehabilitation center of the Bowman Gray School of Medicine of Wake Forest College, Winston-Salem, September 19, 1947.

At the same time, psychiatrists are being called on increasingly to help speed up production in industry, to provide better guidance in schools, to solve the problems of juvenile delinquency—and so on, in just about every walk of life.

The demand for psychiatric counsel and advice is assuming universal proportions. In answer to it, let us make sure we do not respond as "universal specialists"! If we do —by accepting the responsibility for straightening out all the defects in education, and for solving the labor problems in industry and all the political problems of the day, including international relations—we will suffer the consequences.

If we set ourselves up, or should be set up, as the solution to all of man's difficulties, we simply will not be able to deliver the goods —and the fate of those who fail to deliver the goods varies but little, whether "the goods" are psychiatric services or rubber tires. Therein lies the great danger in any public attitude that endows psychiatry with the ability to cure all the ills to which the human body and mind are heir.

The Psychiatrist as a Medical Doctor

The entire world is looking to the present and oncoming generations to produce large-scale, practical ways and means to enrich life and make the stay on this earth more profitable and enjoyable. I am optimistic enough to believe that there will be a better way of life, and I wholeheartedly advocate that those in the field of psychiatry remain alert to see where psychiatrists can be of assistance—not in the role of a universal specialist, not in the role of a second-rate or even a first-rate Houdini, but in the pure and unadorned role of a medical doctor whose contribution is specialized by his knowledge of the human mind.

We must deny any indication that we are equipped to run industry, much less the universe, all by ourselves. The most important thing for the specialist in psychiatry to remember is that he is a medical doctor, and that his contribution as a psychiatrist is primarily that of a medical man. Psychiatrists must think as medical doctors, act as medical doctors, and, above all, *talk* as medical doctors, abandoning terminology and procedures that envelop our specialty with anything reminiscent of the occult or the mysterious.

If we are to measure up to our opportunities and meet our responsibilities in bringing about a better way of life, we must never forget that as psychiatrists we must maintain a close interrelationship with our medical colleagues, and must talk to our patients in simple language that they will understand and remember. It takes no great mind to make simple things complicated, but it takes a very great mind to make complicated things simple.

Qualifications of a Good Psychiatrist

In this effort, as I said before, every psychiatrist must be a well-informed medical doctor with a thorough working knowledge of the human body. He must also be a good psychologist with a practical understanding of psychologic principles. Thirdly, he must be a sociologist, in that he must be a constant student of the social milieu in which his patients have lived and must live in the future. Fourthly, he must be a vocational guidance specialist; and finally, he must be an educator. I believe that psychotherapy, regardless of the form it assumes, is essentially personal tutoring.

One thing more. We have heard a great deal about the desperate need for more psychiatrists, and I take no issue with those who would increase our numbers, as well as the numbers of psychiatric nurses, psychiatrically oriented social workers, and other adjunctive workers. However, I urge equal attention to the *quality* of those who fill our ranks. Quality counts much more than quantity of personnel in giving psychiatry a fighting chance in its attack on mental illness.

Conclusion

If we base our reasoning on those simple concepts of our responsibility, and diligently apply ourselves in uncompromising scientific efforts, the greatest of all public health problems—that of nervous and mental diseases—will yield before the attack of the fine young medical minds, with their high degree of intellectual curiosity, that are now coming into our field.

The challenge to brawn of a century ago is now generally accepted as having changed to a challenge to brains. Let us keep our feet on the ground. Let us every day knit psychiatry more closely to the other medical

specialties, build it more firmly on scientific medical facts, and make each psychiatrist an individually responsible, serious-minded student and exponent of those facts. On those facts the truly brilliant future of psychiatry rests.

HEMOGLOBINURIC OR TUBULAR NEPHROSIS (ACUTE PARENCHYMATOUS NEPHRITIS)

VIRGIL H. MOON, M.D.*

PHILADELPHIA

This presentation originally was planned as a clinicopathologic conference on *hemoglobinuric* or *tubular nephrosis.* On critical examination of our material—didactic, clinical and pathologic—it became evident that another form of presentation is better suited to this subject. Accordingly, your attention is invited to an important syndrome not adequately portrayed in current medical literature. A resume' of its nature and pathogenesis will be followed in this same issue by an article dealing with clinical considerations.

Clinical and Pathologic Descriptions

Internists and pathologists of the previous medical epoch described a form of renal disease which is not discussed in recent textbooks. Osler, in the first edition (1892) of his PRINCIPLES AND PRACTICE OF MEDICINE, described "acute Bright's disease" caused by the action of toxic agents upon the kidneys. This condition resulted from poisons, such as turpentine, phenol, and potassium chlorate; from severe infections, such as septicemia, scarlet fever, measles, diphtheria, malaria, yellow fever and others; from toxic effects of unknown origin, such as the toxemias of pregnancy; and from the effects of burns of the skin. Later editions added trauma and extensive surgical procedures to the etiologic conditions.

Osler gave these urinary changes as signs of this condition: The urine is suppressed or greatly reduced; the quantity may be 4 or 5 ounces per day. It is highly colored, dark, smoky or reddish-brown, and it contains blood, albumin and casts; the specific gravity is high — 1.025 or more. These changes are accompanied by uremic symptoms. Progress since Osler's day has added to these signs the progressive retention of nitrogenous waste products in the blood.

Contemporaries of Osler, both internists and pathologists, described this condition under a variety of terms: acute parenchymatous nephritis, acute tubular nephritis, desquamative nephritis, and others. They gave in general the same etiologic conditions listed by Osler. Pathologists were in agreement on the gross and microscopic changes seen in the kidneys. Adami[1] (1909) gave this description of acute parenchymatous nephritis:

> "The kidneys usually are enlarged and edematous. The capsule strips with more than usual ease. The kidney substance is swollen and tends to bulge. The surface is pale and the stellate veins injected. The cortex is swollen, pale and cloudy, presenting a marked contrast to the dark red medulla. In some cases the cortex is somewhat congested. Minute petechial hemorrhages can frequently be seen in the cortical surface and throughout the substance.
>
> "Microscopically the structures chiefly affected are the convoluted tubules. The cells are swollen, cloudy or granular, often vacuolated, and the nuclei stain poorly or not at all. Hyaline or granular casts and droplets of albumin may be seen within the lumina. Often the lining cells have desquamated from the basement membrane. In some cases the tubules contain blood. Hemorrhage into Bowman's capsule is noted in some cases, or it may contain red and white corpuscles, albumin and detritus. Free blood and blood casts may appear in the urine. The interstitial tissue is swollen and edematous and sometimes there is a small amount of round-celled infiltration and a deposit of fibrin in the interstitial stroma. The extent of the lesions varies in different cases. It is sometimes impossible to draw the line between cloudy swelling and this form of nephritis."

In examinations of material from over 200 cases of this syndrome, I have found no important items which should be added to Adami's description.

Some thirty years ago, a new classification and nomenclature of renal diseases was proposed, based upon the work of Volhard and Fahr. For reasons unknown, "parenchymatous nephritis" was not described as an entity, nor was any name suggested for it. This new classification was widely adopted, and "parenchymatous nephritis" was lost from sight. For a time some internists (McCrae, Tice) still described that clinical syndrome, but most physicians of the present time either are ignorant of the condition or have learned of it in the school of experience.

Presented at the Watts Hospital Symposium, Durham, February 11, 1948.

* Professor of Pathology, Jefferson Medical College, Philadelphia, Pennsylvania.

1. Adami, J. G., and Nicholls, A. G.: Principles of Pathology, Philadelphia, Lea & Febiger, 1909, v. 2, pp. 740-743.

Relation to Shock

My interest in this subject was aroused incidentally. In the course of studies on the pathology of shock[2], it was noted that the kidneys regularly showed extensive tubular degeneration, ranging to necrosis. They presented the complete picture described by Adami and his contemporaries. The clinical findings in such cases had included urinary suppression and dark or pigmented urine of high specific gravity containing albumin, epithelial cells, casts and debris, and usually erythrocytes and hemoglobin pigment as well. These features were as described by Osler. They accompanied a progressive increase in blood urea leading, in severe cases, to death by uremia.

Our studies have shown that the occurrence of shock is not limited to severe injuries, burns, and the aftermath of extensive surgery. This finding now is widely supported. Reviewing the subject of shock, Harkins[3] listed more than thirty conditions in which it may occur. These included extensive surgery; trauma; burning; freezing; radiation burns; sunburn; heat stroke; asphyxia; thrombosis; intestinal strangulation; bile peritonitis; perforated gastric ulcer; pancreatitis; various poisons such as bichloride of mercury, arsenicals, phenol, barbiturates and others; snake venoms; anaphylaxis; transfusion reactions; systemic diseases such as diabetic coma, eclampsia, and toxic jaundice; severe infections such as cholera, diphtheria, pneumonia, and peritonitis; gas gangrene; and various anesthetic agents. I have verified the occurrence of shock in each of these conditions and in others not mentioned.

Shock is a form of circulatory failure which occurs in varying degrees. When severe or profound, it tends to progress inexorably to death; when it is slight or mild, recovery usually follows. When shock is of intermediate or sublethal degree, the patient may live for several days until death occurs as a result of renal failure or terminal pneumonia.

This is the degree of shock which regularly is accompanied by the clinical signs of renal disturbances described by our predecessors in medicine and in pathology. It arises not only in the conditions mentioned by Osler, but in numerous others. As a cause for uremic manifestations, it outranks all others. Deaths from renal insufficiency are due to this cause more frequently than to any other form of renal disorder. Much confusion has arisen as a result of omitting this syndrome from consideration. Many authors have reported deaths from renal failure of this type, not recognizing its relationship to shock and to this form of nephropathy with which our predecessors were familiar.

For example, the "liver deaths" or deaths from the "hepatorenal syndrome" known to surgeons are instances of sublethal shock with renal failure resulting from extensive abdominal operations or from injury to the liver. The "crush syndrome" which Bywaters[4] described is sublethal shock resulting from ischemic necrosis caused by prolonged pressure upon muscles. Bell[5] discussed as "clinical acute nephritis" numerous instances of uremic death not due to glomerular nephritis nor to other organic disease of the kidneys. His observations were made in cases of major surgical procedures, trauma plus infection, barbiturate poisoning, icterus gravis, diabetic coma, pancreatitis, ileus, streptococcal cellulitis, cholangitis, and transfusions with incompatible blood. Shock was recognized clinically in some of these cases.

Recently Lucke'[6] reported observations on "the lower nephron syndrome." He cited more than 400 cases from material studied in the Army Institute of Pathology. These resulted from battle wounds, crush injuries, surgical operations, burns, transfusion reactions, infections, sulfonamide intoxication, poisons, heat stroke, malaria, eclampsia, pancreatitis, and shock from other causes. The clinical signs were oliguria; dark or bloody urine containing pigment, albumin and casts; and progressive azotemia, hypertension, edema and uremia. The pathologic findings in the kidneys were those described by Adami with one important exception: On microscopic study, Lucke' found the degen-

2. (a) Moon, V. H., and Kennedy, P. J.: The Pathology of Shock, Arch. Path. 14:360-371 (Sept.) 1932; (b) Moon, V. H.: Shock: Its Dynamics, Occurrence and Management, Philadelphia, Lea & Febiger, 1942, ch. 9 and 15.
3. Harkins, H. N.: Recent Advances in the Study and Management of Traumatic Shock, Surgery 9:450 (March) 1941.
4. Bywaters, E. G. L.: Effects on Kidney of Limb Compression, Brit. M. J. 2:884 (Dec. 20) 1941.
5. Bell, E. T.: (a) Pathology and Pathogenesis of Clinical Acute Nephritis, Am. J. Path. 13:497-552 (July) 1937; (b) Renal Diseases, Philadelphia, Lea & Febiger, 1946, pp. 254-264.
6. Lucké, B.: Lower Nephron Nephrosis, Mil. Surgeon 99: 371-396 (Nov.) 1946.

eration and necrosis sharply limited to the lower segment of the tubules. Others have reported equal or greater involvement of the upper portion.

More recently, Mallory[7] described this condition as "hemoglobinuric nephrosis." Cases of shock from wounds, burns, heat stroke, crush syndrome, high altitude anoxia, transfusion reactions, and other causes were studied in army hospitals. He stated that the first effect of shock upon renal function was oliguria, followed quickly by retention of nitrogenous wastes. Constant features were oliguria, azotemia, pigment and albumin in an acid urine, and fixation of specific gravity at a low level. In the last particular, his data differ from those of others, who report a concentrated urine of high specific gravity. The severity of the renal deficiency was proportional, in most cases, to the degree of shock. In severe shock, fatal uremia developed in 33 per cent of the cases; after mild shock the mortality was 11 per cent. Mallory described histologic details, but did not stress degeneration of the convoluted tubules, as other writers have. His report supplies additional data confirming the occurrence and the characteristic features of this syndrome.

Case Reports

A few illustrative cases will be cited:

Case 1

Resection of a rectal carcinoma, performed under spinal anesthesia on a man aged 59, "was followed immediately by marked shock from which he did not entirely recover." Repeated transfusions of blood and of plasma did not restore circulatory efficiency. The blood pressure fluctuated between 60 and 100 systolic. Urination was almost completely suppressed. The nonprotein nitrogen and creatinine rose progressively, and death occurred on the fourth day. The clinical diagnosis was uremia secondary to severe postoperative shock.

The kidneys weighed 210 and 190 Gm.; the subcapsular vessels were engorged; the cortex was thickened, opaque, and pink. The tubular epithelium showed granular degeneration, decided vacuolization, and beginning necrosis, which involved chiefly the upper segment. The tubules contained debris and desquamated epithelium, and there was moderate edema of the stroma.

Case 2

This young man's clothing was saturated with gasoline and ignited. He sustained first, second, and third degree burns of the entire body. Treatment for shock with morphine, plasma, and dextrose-saline injections was begun at once; the burned areas were debrided, and pressure dressings were applied. The urine was scanty and dark; it contained albumin, red blood cells, and blood casts. On the fourth day the total output was only 200 cc. of coffee-colored urine. No chemical determinations were made. Death occurred at the end of the fourth day.

The visceral findings at necropsy were those usually seen after death from burns. The kidneys weighed 200 and 240 Gm. respectively; they were soft, swollen, and grayish pink; the cortical markings were obscure, and the superficial vessels were engorged. Amorphous material was present in the capsular spaces. The tubular epithelium showed advanced degenerative changes; it was low cuboidal in form, and the lumina contained debris and casts consisting of red blood cells, hyaline, and dark brown pigment.

It is commonly known that severe *infections* of various types often disturb renal function. This effect seems to depend on the severity rather than on the type of the infection. Numerous accounts of febrile albuminuria may be found; only occasionally did the authors of such articles recognize a relationship to shock. The following case is an example.

Case 3

A severe illness with moderate leukocytosis and a temperature of 103 to 104 F. was treated with sulfonamide drugs and penicillin without effect. The patient's blood pressure declined to 80 systolic, 60 diastolic. He became anuric, and uremic crystals covered the cutaneous surfaces. On the eleventh day of illness the nonprotein nitrogen rose to 288 mg. and the creatinine to 6.6 mg. per 100 cc. of blood. He died in coma on the twelfth day. From the character of the illness and the postmortem observations a provisional diagnosis of systemic tularemia was made.

The necropsy revealed numerous scattered nodules in the lungs and spleen. These contained necrotic areas, fibrin, and monocytic infiltration, such as occur in tularemic inflammation. The spleen and liver were enlarged; the viscera were hyperemic. The kidneys were moderately swollen and contained capillary hemorrhages. There was extensive degeneration and necrosis, especially severe in the proximal portion of the convoluted tubules. The lumina contained debris and casts, both hyaline and granular. These features were of the same order as those seen in renal deficiency from other causes.

It is well known that either *hyperthermia* or *heat stroke* may cause circulatory collapse; in some instances the subject may survive several days in a state of sublethal shock accompanied by renal deficiency. The following case report is an instance.

Case 4

Circulatory collapse developed in this man after six hours at 107 F. in hyperthermic treatment for gonorrhea. On his removal from the cabinet the blood pressure fell to 50 systolic, 30 diastolic. The pulse rate was 156, and the urea nitrogen of the blood was 22 mg. per 100 cc. Oliguria and albuminuria were present, and the urine contained erythrocytes and casts. Supportive treatment was ineffective; the circulatory deficiency continued, and the blood urea nitrogen rose to 77 mg. per 100 cc. Death

7. Mallory, T. B.: Hemoglobinuric Nephrosis in Traumatic Shock, Am. J. Clin. Path. 17:427-443 (June) 1947.

came ninety-eight hours after the therapeutic hyperthermia.

As is usual in such cases, the visceral findings were those characteristic of secondary shock. The kidneys, weighing 240 and 250 Gm., were swollen and reddish brown. The cortical markings were obscure, and the pelvic lining contained petechial hemorrhages. Microscopically, both cortex and medulla were hyperemic. The tubular epithelium was severely degenerated; the lumina were wide and contained debris and desquamated cells; the collecting tubules contained casts and many erythrocytes. The stroma was edematous.

In military aviation at *high altitudes*, circulatory collapse resembling surgical shock often developed even though oxygen was adequately supplied. "The most dangerous aspects are seen after a latent period of one to six hours, resulting in a full-blown vicious circle of peripheral circulatory insufficiency, tissue anoxia and marked hemoconcentration."[8] I had the opportunity to examine the records and material in 5 such cases. The following is a typical example.

Case 5

This man was undergoing a test in the low-pressure chamber at a relative altitude of 38,000 feet, when pains and distress developed. After gradual recompression he seemed drowsy and incoherent, and was put to bed immediately. The pulse rate then was 80, and the blood pressure 110 systolic, 80 diastolic. Severe circulatory deficiency developed four hours later. The pulse could not be obtained; the hematocrit reading had risen to 64.9; the patient was sweating, and apparently was in profound shock. He was given plasma and isotonic solution of sodium chloride intravenously, and oxygen by inhalation. Severe hyperpyrexia developed, like that which accompanies heat stroke; the rectal temperature rose to 108.4 F. The urine was scanty and contained albumin (4 plus). No blood chemical tests were made. Despite the administration of intravenous fluids and other measures, the circulatory deficiency persisted; death came forty-five hours after the patient's removal from the low-pressure chamber.

The necropsy observations were the same as those seen in secondary shock from other causes. Microscopic examination of the kidneys revealed the identical pattern of changes seen in the crush syndrome or after transfusion with incompatible blood. It is significant that numerous pigmented casts were found, although no transfusions and no therapy with sulfonamide compounds or other drugs had been given.

Space is lacking for details of other instances. These would include intestinal obstruction, peritonitis, perforation of viscera, coronary occlusion, radiation sickness, toxic jaundice, transfusion reaction, anaphylaxis, poisoning with various chemicals, reaction to arsenical therapy, overdoses of sedatives and of sulfonamide drugs, diabetic crises and others. Each of these conditions, if its severity is maximal, will result in the syndrome of shock leading to death. Each of them, when less severe, may cause the syndrome of uremia leading to delayed death. The morphologic changes in the kidneys are of the same pattern, regardless of the causative condition.

8. Physiology of Flight, Air Forces Manual 25-2, United States War Department, Army Air Forces, 1945, pp. 17 and 36.

Pathogenesis

The exact mechanism by which renal failure develops under the conditions mentioned has not yet been shown, although several explanations have been advanced. It was suggested that the collecting tubules become plugged with pigment, cells, debris and casts; that these obstruct mechanically the outflow of urine from kidneys otherwise functioning normally. This explanation is not consistent with the fact that only a relatively small proportion of the tubules are obstructed. I have never seen an instance in which 20 per cent of the tubules were plugged. The remaining 80 per cent with unobstructed outflow should be sufficient to prevent death by urinary retention.

Some have emphasized that kidneys cannot form urine when the blood pressure is low. This explanation is sound physiologically when the arterial pressure is abnormally and continuously reduced. In many instances, however, the clinical records show that the blood pressure was maintained at adequate levels by intravenous fluids, transfusions, and other means. In such cases, adequate arterial pressure did not prevent fatal renal insufficiency.

It has been noted that hemoconcentration often is marked during shock, and that the glomeruli cannot produce a filtrate from abnormally concentrated blood. In many cases, however, hemoconcentration either was not present or had been corrected by fluids given intravenously; yet renal insufficiency progressed to a fatal termination.

Lack of oxygen—anoxia—has now been accepted as a regular feature of shock. This may develop as a systemic condition, or it may affect chiefly the kidneys as a result of reduced arterial blood flow through them. In either case, renal function cannot be maintained in the absence of an adequate supply of oxygen. Lack of oxygen is probably an important factor in renal failure complicating sublethal shock.

Years ago, Richards[9] and his associates

9. Richards, A. N.: Direct Observations of Change in Function of the Renal Tubule, Caused by Certain Poisons, Tr. A. Am. Physicians 44:64-67, 1929.

showed that poisons, such as bichloride of mercury and others, caused degeneration and necrosis of the tubular epithelium. This development was followed by suppression of urine and by death. They found that degeneration of the epithelium reduced its function of selective absorption by which the glomerular filtrate is concentrated. The epithelium, if damaged, allowed the entire filtrate to be resorbed into the circulation by a process of diffusion. This resorption caused marked oliguria or anuria by interrupting the process by which the urine is formed.

Probably the two last mechanisms are most important in this syndrome. Anoxia of itself will reduce renal function directly. It will also cause degeneration of tubular epithelium, bringing into action the process described by Richards.

Terminology

Confusion as to terminology will continue until common usage sanctions a suitable name designating this syndrome. The term *toxic nephrosis* is open to the objection that, in many instances, no toxic agent is implicated. In shock from anaphylaxis, from transfusion with incompatible blood, from therapeutic hyperthermia or heat stroke, from low atmospheric pressure, from anoxia or asphyxia, no toxic effects seem probable. The term *hemoglobinuric nephrosis* is free from objection and has the merit of designating an important sign of the condition —hemoglobinuria. *Tubular nephrosis* likewise is acceptable, suggesting the chief pathologic lesion. The term *parenchymatous nephritis* has historic significance, as it was applied to this syndrome by our predecessors. Unfortunately it implies an *inflammatory* feature which is lacking in this form of renal disturbance.

Summary

A form of renal insufficiency which is associated with a sublethal degree of shock resulting from various causes produces uremic deaths more frequently than do organic diseases of the kidneys.

Renal failure probably is due to degeneration and necrosis of tubular epithelium, which may result from reduced arterial circulation, anoxia, or toxic agents. Such degeneration interrupts the formation of urine.

TUBULAR NEPHROSIS

Clinical Considerations

GARFIELD G. DUNCAN, M.D.*

PHILADELPHIA

The important clinical considerations concerning the so-called extrarenal azotemia or tubular nephrosis syndrome are its *cause*, its *recognition*, its *prevention*, and its *treatment*. The mechanisms set in motion by the various conditions associated with shock have been considered by Dr. Moon. An understanding of these mechanisms is essential as a basis for the clinical management of this syndrome.

Recognition

Oliguria (or anuria), azotemia, hypertension, and the passing of granular and pigmented casts in the urine following clinical crises are characteristics of acute tubular nephrosis. The oliguria may be transitory, especially in the cases of dehydration. In such cases it is indicative of an effective protective mechanism thrown into operation to prevent reduction of blood volume. As the urine volume falls below 500 cc., however, the danger of grave disturbances in renal function increases.

The onset of anuria may be sudden, as following peripheral vascular collapse during surgery; or oliguria may develop gradually following the precipitating crisis. In the latter instance, the urine, in the early stages, has a high specific gravity, and contains red blood cells, and granular and pigmented casts. As the oliguria persists, in spite of an increased fluid intake, the specific gravity of the highly acid urine falls to relatively fixed levels—between 1.008 and 1.011. Concomitantly the blood pressure, previously subnormal, increases to 160 systolic, 100 diastolic or thereabouts, and the urea nitrogen value of the blood increases from the normal of 12-15 mg. per 100 cc. to uremic levels. Lucke[1] has estimated the mortality rate in such cases to be approximately 90 per cent. In those cases in which recovery is achieved,

Presented at the Watts Hospital Symposium, Durham, February 11, 1948.

From the Benjamin Franklin Clinic of the Pennsylvania Hospital, Philadelphia, Pennsylvania.

* Clinical Professor of Medicine, Jefferson Medical College, Philadelphia, Pennsylvania.

1. Lucké, B.: Lower Nephron Nephrosis, Mil. Surgeon 99: 371-396 (Nov.) 1946.

the secretion of urine is resumed gradually, after variable periods of anuria. The blood urea nitrogen values return to normal over a period of five to fifteen days, and normal blood pressure readings are restored. The specific gravity of the urine remains fixed at the low level for weeks or months, and sometimes it is several years before it returns to normal. That the specific gravity usually does return to normal, even after a great lapse of time, is an indication of what can be expected in the way of repair of tubular damage; in contrast, predominant glomerular damage results in permanent impairment of function.

Edema is a variable feature and is often related to the therapy employed.

Cause

In considering the disturbed physiology of acute tubular nephrosis we recall that Trueta and his coworkers[2] discovered that, during experimental shock, the cortical glomeruli of the kidney are by-passed by a short-circuiting of the blood flow *via* a medullary route. The blood flows through the juxta-medullary glomeruli, their efferent vessels, and their respective vasa recta to the interlobular veins. This hypothesis, so convincingly presented, indicates that oliguria and anuria result from neurogenic vascular spasm, which prevents blood from reaching the cortical glomeruli in sufficient quantity to permit a normal amount of urine to be excreted. If this is the phenomenon responsible for the development of so-called extrarenal azotemia—and it appears quite probable that it is—we shall be obliged to abandon the theory of complete tubular absorption in anuric patients. It is quite feasible too, and in accord with Trueta's observations, that the correction of anuria and oliguria coincides with the return of an adequate circulation through the cortical glomeruli, but that the tubules, having suffered from anoxia, fail in their more highly specialized function of concentrating the urine.

Prevention

Many circumstances influence the occurrence of tubular nephrosis. In my experience most of the fatalities have occurred in postoperative cases, and, in some, transfusions of whole blood had been given. The importance of proper matching of blood need hardly be mentioned. I have never seen a case of tubular nephrosis in patients receiving sulfonamides, if two simple rules were observed: (1) the patient must pass at least 1500 cc. of urine per day, and (2) at least one specimen in each twenty-four hours must be alkaline in reaction.

The prevention of shock is not always possible. Patients are often in severe shock when first seen by the physician. Appropriate attention to the nutritional status makes it possible to reduce remarkably the susceptibility to shock, and hence to tubular nephrosis, in patients undergoing surgery. When the prevalent practice of prescribing or permitting semi-starvation during acute and chronic infections is abandoned, further progress in this direction can be expected. Lyons[3], Gregersen[4], Evans[5], and Ling and Sprinz[6] have found a definite relationship between loss of weight and susceptibility to surgical shock. Lyons found that patients who lost weight suffered, at the same time, a decrease in blood volume which masked reductions in the total circulating hemoglobin, red blood cells, and protein. Hence, in such patients, normal red cell counts and normal protein and hemoglobin concentrations should be accepted as such only if the blood volume is found to be normal also. Operative procedures should be done *after, not before,* the blood volume is corrected by transfusions of whole blood.

Much harm has been done by severely restricting protein in the treatments for cardiovascular and renal diseases, for acute and chronic diseases of the liver, and for surgical conditions. All are agreed that starvation for protracted periods increases the surgical risk. Protein and calorie depletion played a prominent part in Lyons' and Ling's patients, many of whom were malnourished soldiers with chronic suppurating wounds. Ling and Sprinz found, in these patients, an average reduction in blood volume of 1430

2. (a) Trueta, J. and others: Studies of the Renal Circulation, Charles C. Thomas, Springfield, Ill., 1947; (b) Trueta, J. and others: Renal Pathology in the Light of Recent Neurovascular Studies, Lancet 2:237-238 (Aug. 17) 1946.

3. (a) Lyons, C.: Penicillin Therapy of Surgical Infections in the United States Army, J.A.M.A. 123:1007-1018 (Dec. 18) 1943; (b) Lyons, C. and Mayerson, H. S.: The Surgical Significance of Hemoglobin Deficiency in Protein Depletion, J.A.M.A. 135:9-11 (Sept. 6) 1947.

4. Personal communication to Ling(6).

5. Evans, E. I.: Discussion of Lyons(3b).

6. Ling, W. S. M. and Sprinz, H.: Blood Volume Changes of Available (Thiocyanate) Fluid Space in Soldiers with Chronic Wound Infections and Associated Nutritional Depletion, Am. J. M. Sc., in press.

cc., or 24.2 per cent. Ev[illegible] [5] found that approximately 50 per cen[illegible]f hospitalized patients have reduced blood volumes, and Lyons [3b] has verified this finding in cases of chronic infections, hepatic insufficiency, undernutrition, and malignant tumors.

Patients admitted to hospitals present all phases of undernutrition, which in some cases may be due to bizarre and inadequate therapeutic diets. During the course of pregnancy, lactation, organic disease, and infection, patients are more vulnerable to the untoward effects of undernutrition. Eclampsia is a nutritional problem, and the changes in the kidneys of the eclamptic patient fall into the tubular nephrosis group.

It is established that malnutrition plays an important part in predisposing to surgical shock[2b,5] and eclampsia[7]. It is reasonable to suppose that it may be a factor in the production of tubular nephrosis from other causes. In this connection the results of a nutrition survey made recently at the Pennsylvania Hospital[8] are pertinent. The nutrient intake of each patient on each ward was evaluated for two successive days. All food delivered to each patient and all leftovers were weighed. These studies, which covered the medical, surgical, and obstetric and gynecologic divisions, indicated that hospitalized patients frequently do not eat an adequate diet. Consider the surgical risk of a patient suffering from bronchiectasis whose daily intake was 29 Gm. of protein and 753 calories.

Case Reports

Case 1

A 39-year-old man was admitted to the Pennsylvania Hospital on September 9, 1947, complaining of loss of weight and severe cough of five years' duration, and the expectoration of approximately 8 ounces of purulent sputum daily. He had fever, severe sweats, and leukocytosis. Roentgenograms showed an abscess cavity in the right upper lobe with considerable pulmonary atelectasis.

Some improvement followed penicillin therapy, but he continued to have fever, leukocytosis, and large quantities of purulent sputum. Following three transfusions, 1500 cc. in all, the upper and middle lobes of the right lung were removed on December 18. The upper lobe was atelectatic, with extensive chronic and acute pneumonitis and two cavities. The operation was long and difficult, and when it was nearly over the patient went into severe shock. Three and one half liters of whole blood were given before the blood pressure rose to 100 systolic, 80 diastolic.

7. Tompkins, W. T.: The Significance of Nutritional Deficiency in Pregnancy; A Preliminary Report, J. Internat. Coll. Surgeons 4:147-153 (April) 1941.
8. Duncan, G. G., and Ling, W. S. M.: Some Nutritional Hazards in a General Hospital, Tr. Assoc. Am. Physicians 60:208-217, 1947.

The patient was **anuric** until the third postoperative day, when 15 cc. containing albumin (3 plus), red blood cells, and granular and pigmented casts were excreted. On the fourth day 30 cc. were passed, and on the fifth 210 cc., having a specific gravity of 1.010. On the sixth day the urine volume was 310 cc.; on the eighth day 1540 cc. The twenty-four hour excretion on the ninth day amounted to 1480 cc. with a specific gravity of 1.010, and containing only 1.65 Gm. of sodium chloride and 2.5 Gm. of urea. The urine volume remained satisfactory thereafter, but the specific gravity has remained low—between 1.006 and 1.012.

The blood **urea nitrogen** concentration, which was normal (14 mg. per 100 cc.) before operation, increased steadily to 225 mg. per 100 cc. on the eighth day, when the **creatinine** was 8.2 mg. per 100 cc. Thereafter, the values decreased, the creatinine value being 1.7 mg. on the thirteenth day and the blood urea nitrogen 22 mg. one month after operation. The **blood chloride** values were not abnormal. On the third postoperative day the **total base** had increased to 179 milli-equivalents per liter (Sunderman's method), and eighteen hours later to 193. Following mild diarrhea it fell to 179 on the fourth day, but increased to 191 on the fifth day. Again, following looseness of the bowels, the total base fell to 160 on the sixth postoperative day. The carbon-dioxide **combining power** did not decrease with the reduction in the total base. The values were 33 volumes per cent on the fourth, and 36 and 37 volumes per cent on the sixth postoperative day, when the total base had been reduced to 160 milli-equivalents. On the eleventh day the carbon dioxide combining power was 39 volumes per cent, and the total base 152 milli-equivalents per liter.

The **specific gravity of the blood** following the administration of heparin was reduced to 1.051 on the evening of the second postoperative day, after extensive hydration. On the third day the following values were noted: 2 a.m., 1.055; 11 a.m., 1.056; and 1:30 p.m. 1.050. The specific gravity increased to 1.060 on the sixth day, but with increased hydration, values of 1.049 and 1.046 were noted on the sixth and eighth days respectively.

The **blood volume** on the evening of the second day was 5490 cc., as compared with the calculated normal of 5600 cc., and the hematocrit was 51 per cent. The plasma volume was 2691 cc. (normal 2744 cc.). On the third day the blood volume had decreased to 4110 cc. and the plasma to 2401 cc. It was at that time that a few crackling rales were noted at the base of the left lung, and on the following day edema of the extremities was detected. Following the administration of 100 Gm. of concentrated human albumin, the blood volume was restored to 5500 cc. on the fifth day.

The **serum protein** values were at no time significantly abnormal. The **bilirubin value** on the second postoperative day was 2.7 mg. per 100 cc.

The clinical condition of this patient improved for a short time—two days—after the state of shock had been corrected, but thereafter he became increasingly drowsy and irrational until the kidney function was restored. Hiccoughs and nausea were troublesome. The **blood pressure** prior to operation was normal; following the period of shock it gradually increased to 160 systolic, 100 diastolic on the third day, and subsequently fell gradually to normal levels.

In summary, this patient had suffered from a chronic infection for several years and had become malnourished, so that he was a good candidate for so-called "chronic

shock." Acute shock, with evidence of hemolysis following large transfusions of whole blood, predisposed to the rapid development of acute tubular nephrosis.

Case 2

A white woman, aged 37 years, was admitted to the Pennsylvania Hospital on November 10, 1947, eight days after operation for appendicitis in another hospital. After receiving a transfusion of whole blood she had voided no urine for five days, and had become comatose. On the day of admission she had regained consciousness, but was drowsy, and hiccoughs were troublesome. One hundred and fifty cc. of bloody urine, having a specific gravity of 1.008 and containing 3.5 Gm. of urea, were obtained by catheter on the day of admission. Increasing amounts were voided on successive days, as follows: 620 cc., 750 cc., 1435 cc., and 1620 cc. All specimens had a low specific gravity. The patient had a low-grade fever, a purulent discharge from her appendectomy wound, and leukocytosis.

On the second hospital day a pulmonary embolism occurred, and for several hours the prognosis appeared hopeless. Although heparin therapy was begun at once, the coagulation time was allowed to fall below twenty minutes, and on the fourth hospital day another pulmonary embolism occurred. At this time a rapidly progressive edema of the vulva was detected and a diagnosis of thrombophlebitis of the pelvic veins was made.

Classic signs of cardiac failure, which yielded to digitalis therapy, were noted on the sixth day. Following the development of a pelvic abscess, which ruptured into the vagina, her recovery was uneventful, except for an attack of pyelonephritis due to Bacillus coli. She was discharged on December 7, after twenty-seven days of hospitalization.

The **blood pressure** during the oliguric phase was as high as 156 systolic, 110 diastolic, but before discharge, levels of 140 systolic, 70 diastolic and 132 systolic, 78 diastolic were noted. The **specific gravity** of the urine was low, 1.010-1.012, during the oliguric phase, but readings of 1.020 and 1.021 were recorded prior to discharge. The **blood urea** fell steadily from 133 mg. per 100 cc. on admission to 21 before discharge, and the **creatinine** from 7.5 to 1.4 mg. The **carbon dioxide combining power** was 37 volumes per cent (16.6 milli-equivalents) on admission. On the following day it was 35 (15.7 milli-equivalents), and twenty-four hours later it was 71 volumes per cent (32 milli-equivalents). The **total blood proteins** varied from 4.8 to 5.6 per cent. The total **blood volume** on the second day in the hospital was 5,633 cc. (normal approximately 5,500); the **plasma volume** was 3,380 (normal approximately 3,235), and the **hematocrit** 40 per cent. The blood **sodium chloride** values were low—476 mg. (81 milli-equivalents per 100 cc.), and 484 mg. (83 milli-equivalents)—during the first two days in the hospital.

Treatment

No two patients suffering from the azotemia of acute tubular nephrosis will be treated in exactly the same manner. Each of the several combinations of chemical abnormalities will indicate appropriate therapy (table 1).

The aim is to maintain a suitable, constant environment to the vital tissue cells until healing occurs. To accomplish this requires a team—an internist, a chemist, a genito-urinary surgeon to rule out extrarenal obstruction, and members of the resident staff, one of whom must devote much of his time to the patient during the critical phase, the end of which will be marked by the secretion of a normal volume of urine. The team should begin coordinated operations as soon as oliguria is noted, and *not* after the patient has been anuric for several days and overhydrated in attempts to produce urine.

Laboratory determinations done in batteries to give comparative simultaneous values are imperative for best results. The scattering of determinations leads to confusion and danger.

Table 1

Immediate Therapy Indicated by a Battery of Tests in One Case of Acute Tubular Nephrosis

	Normal Values	*Actual Values*	*Treatment Indicated*	*Amount Administered*
Specific gravity of blood	1.052-1.057	1.039†	*Suspension of red blood cells in saline	650 cc.
Hematocrit (%)	43	25*		
Blood volume (cc.)	4845	4537*		
Plasma volume (cc.)	2565	3630		
R. b. c. volume (cc.)	2280	907*	†Concentrated human albumin (salt free)	75 Gm. (300 cc.)
CO_2 combining power (vol. %)	55-65	25‡		
Chlorides (milli-eq. per liter)	98-103	103.8		
Plasma proteins (Gm. per 100 cc.)	7.1	4.9†		
Plasma albumin (Gm. per 100 cc.)	4.1	2.9†		
Total base (milli-eq. per liter)	140	132‡	‡Sodium lactate solution	400 cc.
Ph	7.3-7.4	7.2‡		
Urea N (mg. per 100 cc.)	10-15	176		
Creatinine (mg. per 100 cc.)	1-1.5	7.02		

The studies considered essential in guiding therapy are:

a. Hemoglobin
b. Specific gravity of whole blood
c. Hematocrit
d. Blood volume
e. Carbon dioxide combining power
f. Chlorides
g. Plasma proteins and albumin-globulin ratio (one determination)
h. Total base

This battery of tests is indicated at the outset of treatment and, with the exception of the albumin-globulin ratio, is repeated in twelve hours, and thereafter at twenty-four hour intervals during the anuric phase.

Urea and creatinine concentrations will indicate the course of the disease, but provide little help in therapy.

Agents employed to maintain water and electrolyte balance

The agents employed in controlling water and electrolyte balance until kidney function is resumed are indicated as follows: When the specific gravity of the blood exceeds normal (1.055), the hematocrit value is above 45 per cent, and the blood volume is below the calculated normal, *5 per cent glucose in distilled water* is given until normal values are restored. Varying circumstances will indicate the addition of certain constituents without changing the desired volume of fluid to be administered. If the total base is decreased and the blood volume is normal, *physiologic normal saline*—or, perhaps better, Ringer's solution—is given in quantities not exceeding the amount necessary to bring the total base to normal (ordinarily not in excess of 500 cc.). If the carbon dioxide combining power as well as the total base is depressed, sodium-lactate solution is given to restore satisfactory values.

Plasma may be used to advantage when the plasma protein is depleted and the total base is depressed, although the greater concentration of protein in *concentrated human albumin*, and its freedom from salt, make this the preparation of choice in most cases.

The total amount of fluid to be administered will of necessity vary with the state of hydration and the fluid losses. Overhydration readily leads to pulmonary edema, and doubtless increases the swelling of the kidneys. It is safe to give sufficient fluid, including glucose, albumin, and the indicated electrolytes, to raise the blood volume to normal. If the blood volume is normal, these vital agents, as indicated, are incorporated in the fluid which is given to replace insensible water loss (loss by way of the lungs, skin, feces, and saliva). This amounts to approximately 1200 cc. per day. To this amount is added sufficient fluid to replace losses by vomiting and diarrhea. The reason for delaying the time-honored flushing of the kidneys is that dilution of the metabolic agents slows up metabolic processes which are basic to healing.

Dr. Olive Hoffman, a member of our team to deal with anuric patients, advocates *catharsis* if the blood volume, total base, and Ph of the blood are elevated. She warns, however, that this is a two-edged sword, and, because of the danger of untoward shifts in the electrolyte balance, should not be employed unless adequate and reliable laboratory data are available. Catharsis is absolutely contraindicated if the total base and Ph are both reduced.

Heparin

In the last 4 patients I have treated for this syndrome I have used heparin. As all have recovered, it seems to have done no harm, and I suspect that it has been beneficial. I make no claims for it, but will present my reasons for using it: (1) Because of the renal stasis and hematuria, and the thromboplastin present in the urine[9], any therapy which might halt the formation of casts of heme compounds in the renal tubules seems advisable; (2) it was considered probable that the thrombophlebitis occasionally observed in the kidneys of these patients might have a vasospastic effect on the arteries and capillaries similar to that seen in thrombophlebitis of an extremity. For these two probably insufficient reasons we are using heparin in these cases at the Pennsylvania Hospital. It is begun as early as possible—100 mg. being given intravenously at once, and 200 mg. introduced in 1000 cc. of 5 per cent glucose at the rate of eight drops per minute initially. The rate of administration is adjusted to maintain a coagulation time of between thirty and fifty minutes. Coagulation times are determined at four-hour intervals, day and night. It is considered safe to use heparin forty-eight hours

9. Tocantins, L. M., and Lindquist, J. N.: Thromboplastic Activity of Urine, Proc. Soc. Exper. Biol. & Med. 65:44-49 (May) 1947.

after surgery. We have had protamine on hand to halt the action of heparin in case of hemorrhage, but have not had to use it. Heparin is discontinued when urine reappears, or when the degree of oliguria is reduced.

Diet

The diet given to the 2 patients whose case reports have been presented in this paper violates time-honored rules. The same violations were observed in 4 other cases—in all, 6 consecutive patients, all of whom recovered. Very small meals were served, nearly all of the prescribed diet being given in a concentrated liquid formula containing salt-free milk, eggs, Protenum and Essenamine. I suspect that the high protein intake was of significant therapeutic value, and that nourishment given frequently aided in preventing intractable nausea and vomiting. The feedings were taken voluntarily when practicable. Otherwise, they were administered by gavage. Nitrogen retention *per se* is not a contraindication to a high-protein diet.

Correction of overhydration

Maintenance of an efficient circulation is extremely important, as the magnitude of disturbance in the renal circulation keeps ahead of that in the general circulation. Cardiac embarrassment and edema are, I fear, usually the result of overhydration. Studies of the venous pressure and circulation time are of value. Venesection, together with the administration of red blood cells and concentrated human albumin, has been life-saving in cases where the blood volume was above normal, with a hematocrit of 25 per cent and a plasma albumin of 2.9 per cent. Digitalis therapy is used when indicated. The partial neutralizing effect of digitalis on heparin increases the difficulties in keeping a uniform coagulation time.

Other measures

Sympathetic nerve block[10] by spinal anesthesia has been effective in restoring renal function in cases of reflex anuria. *Peritoneal lavage*[11] is an emergency measure which offers some promise in desperate cases. I have had no experience with it, nor with Kolff's artificial kidney[12], which has considerable merit with little risk if its use is guided by adequate laboratory data. The use of *extra-abdominal intestinal loops* for diuretic purposes has been tried, but has less to support it as a method of therapy than any of the infrequently used measures mentioned above. *Renal decapsulation* may have merit, but this fact is difficult to prove. When all else fails, there can be no legitimate objection to this procedure.

10. Hingson, R. A., and others: New Horizons in Therapeutic Nerve Block in the Treatment of Vascular and Renal Emergencies with Continuous Caudal and Continuous Spinal Analgesia and Anesthesia, South. Surgeon, 13:580-609 (Aug.) 1947.
11. Abbott, W. E. and Shea, P.: Treatment of Temporary Renal Insufficiency (Uremia) by Peritoneal Lavage, Am. J. M. Sc. 211:312-319 (March) 1946.

Conclusion

Tubular nephrosis is a common disorder with a high mortality rate. Death usually results, not from uremia, but rather from circulatory disorders precipitated by overhydration, which is preventable if therapy is guided by adequate laboratory studies. Administration of fluid is guided by blood-volume studies. Usually it is restricted or not above basic requirements in the early phases of treatment.

A high protein intake is desirable, as is the judicious use of whole blood, red blood cells, plasma, concentrated human albumin, normal saline, and sodium-lactate solution when specific indications arise. Heparin therapy, until the oliguria begins to improve or anuria is corrected, is suggested.

Reasons have been presented for believing that, with adequate facilities, most of these patients can be saved if the oliguria is recognized early and if overhydration is avoided.

12. Kidney Substitutes, Special Article, Lancet 2:726-727 (Nov. 16) 1946.

Gastric and duodenal ulcer.—We have watched these diseases—which were at one time, judging by earlier records of the clinic and the dead-house, relatively rare—becoming in the course of two generations two of the most common of all. Their incidence is still rising. If the combined total of living persons who have or have had an ulcer in America and the British Isles could be computed, they would be found to number not tens or hundreds of thousands but millions. Every endeavour, orthodox and unorthodox, medical and surgical, has been made to discover a cure, but with no very outstanding or encouraging results. Animal and laboratory experiments have carried us but a very little way in the assessment of cause. And yet the causes (for ulcer will probably find its place among the "multiple stress" diseases) must have been developing contemporaneously with the rising incidence. A disease which was once rare can become so again. Until we study the victims of the disease at first hand and in relation to their work, their total occupational experience, their communities, their food, their habits and anxieties, and their innate predispositions, are we likely to obtain the answer to our question? —John A. Ryle: Social Pathology and the New Era in Medicine, Bull. New York Acad. Med. 23:327 (June) 1947.

LUNG ABSCESS

A Review of Forty-Nine Cases

FELDA HIGHTOWER, M.D.
and
H. H. BRADSHAW, M.D.
WINSTON-SALEM

Forty-nine cases of lung abscess were treated on the surgical service of the North Carolina Baptist Hospital from 1941 to 1946, inclusive. The literature is replete with writings on this subject, almost all of which give high mortality rates regardless of the type of therapy employed. In our opinion the immediate operative and postoperative mortality is greater with lung abscess than with any other common pulmonary condition, including carcinoma. The availability of chemotherapeutic and antibiotic agents, the widespread use of supportive blood transfusions, and the great improvements made in the technique of thoracic surgery have failed to reduce appreciably the mortality from pulmonary abscess. In an effort to throw more light on this problem, we have undertaken an analysis of the cases we have treated.

Age, Sex, and Occupation of Patients

The ages of our patients ranged from 4 to 54 years, the average being 32. The age incidence by decades (table 1) is similar to that reported by Woods and McGrath[1] in a review of 71 cases. The highest incidence in our series was in the fifth decade. Some of the textbooks of surgery[2] state that the incidence of the disease is highest in the third decade.

Thirty-three of our patients were men, and 16 were women—a ratio of almost exactly 2 to 1 (table 1). This ratio corresponds closely to those reported by other authors[1,2b].

The occupations of our patients are shown in table 2.

Table 1

Age and Sex of Patients

Age (Years)	*No.*	*Per Cent*
1-9	4	8.2
10-19	8	16.3
20-29	6	12.2
30-39	12	24.5
40-49	14	28.6
50-59	5	10.2
Sex	*No.*	*Per Cent*
Males	33	69.4
Females	16	30.6

Table 2

Occupation of Patients

	No.	*Per Cent*
Child	11	22.4
Housewife	8	16.3
Textile worker	5	10.2
Farmer	5	10.2
Woodworker	3	6.1
Miscellaneous	17	34.7

Predisposing Factors

Table 3 shows the conditions which were believed to be predisposing factors in this series. Pneumonia preceded the development of lung abscess in 14 of the 49 cases, or 28.5 per cent. The respiratory infections (colds, influenza, and pneumonia) considered as a group were believed to be the pathogenic factor in 26 cases. Pneumococcus pneumonia is not thought to be a cause of lung abscess, but there is evidence to support the belief that pneumonia due to other organisms does produce changes in the lung predisposing to lung abscess. Valle[3] reported that 65 per cent of the 244 cases in his series followed pneumonia. Woods and McGrath reported that 33 of their 71 patients (46.4 per cent) developed abscess following pneumonia. D'Ingianni[4], in discussing the pathogenesis of nontuberculous abscesses in children, reported the incidence of pneumonia as 32.2 per cent. He quoted Derges as saying that the most common cause of lung abscess is previous surgery, and that pneumonia is the next most frequent contributing factor.

There were 7 instances of inhalation of foreign bodies in our series. Four patients developed lung abscesses following tonsillectomy and adenoidectomy. Heavy lifting was given as the predisposing factor in 2 cases.

Early Symptoms (Table 4)

Forty-three of our patients (87.7 per

From the Department of Surgery, Bowman Gray School of Medicine of Wake Forest College and the North Carolina Baptist Hospital, Winston-Salem, N. C.

Read before the Section on Surgery, Medical Society of the State of North Carolina, Virginia Beach, Virginia, May 14, 1947.

1. Woods, F. M. and McGrath, E. J.: Management of Pulmonary Abscess, Surgery, 20:187-190 (Aug.) 1946.
2. (a) Babcock, W. and others: Principles and Practice of Surgery, Philadelphia, Lea and Febiger, 1944, p. 836; (b) Hedblom, C. A.: Pulmonary Abscess, in Walters' Practice of Surgery, Hagerstown, Md., W. F. Prior Co., 1947, v. 5, ch. 2, pp. 1 and 2.
3. Valle, A. R.: Lung Abscess: An Analysis of 244 Cases, Surg., Gynec. & Obst. 81:278-286 (Sept.) 1945.
4. D'Ingianni, V.: Nontuberculous Lung Abscesses in Children: Survey of 64 Cases, J. Thoracic Surg. 14:258-264 (June) 1945.

Table 3

Predisposing Factors

	No. Cases	*Per Cent*
Influenza, colds and pneumonia	26	53.1
Aspiration of foreign bodies	7	14.3
Tonsillectomy and adenoidectomy	4	8.2
Other operations	3	6.1
Heavy lifting	2	4.1
Gunshot wound of chest	1	2.0
Amebiasis	1	2.0
Tuberculosis	1	2.0
Carcinoma	1	2.0
Unknown	3	6.1

Table 4

Initial Symptoms

	No. Cases	*Per Cent*
Cough	43	87.7
Foul sputum	22	44.9
Pain	22	44.9
Fever	16	32.6
Hemorrhage	3	6.1

Table 5

Duration of Symptoms

Weeks	*No. Cases*	*Per Cent*
0-6	10	20.4
7-12	12	24.5
13-24	9	18.4
25-48	5	10.2
49 or more	13	26.5

cent) stated that cough was one of their first symptoms. Fever, pain in the chest, or expectoration of foul sputum was the initial symptom in 22 cases (44.9 per cent). Three patients had cough with hemoptysis as the first symptom. Only 3 patients had persistent non-productive cough and fever. Hedblom[2b] and Valle[3] commented on the fact that cough is almost always present in cases of lung abscess.

Duration of Symptoms

The duration of symptoms in these cases ranged from two weeks to twenty years (table 5). Only 10 out of the 49 (20.4 per cent) had been sick less than six weeks. Thus, according to Neuhof's classification[5], 79.6 per cent would be considered as cases of chronic lung abscess. Crimm[6] and Valle[3], on the other hand, do not classify an abscess as chronic until it has existed for more than two months. Brantigan and Looper[7] found the usual classification of abscesses as acute or chronic to be unsatisfactory. They preferred to use the terms "simple or uncomplicated" and "chronic or complicated." While

5. Neuhof, H.: Acute Putrid Abscess of the Lung. Surg., Gynec. & Obst. 80:351-354 (April) 1945.
6. Crimm, P. D.: Lung Abscess. J. Indiana M. A. 39:17-23 (Jan.) 1946.
7. Brantigan, O. C. and Looper, E. A.: The Etiology and Surgical Treatment of Lung Abscess; Importance of Lobectomy. South. M. J. 37:199-207 (April) 1944.

Table 6

Location of Abscess

	No. Cases	*Per Cent*
Single lobe involved		
Right lower	11	22.4
Left lower	6	12.2
Left upper	4	8.2
Right upper	2	4.1
Right middle	1	2.1
Multiple lobes involved		
Right upper, middle, and lower	11	22.4
Right upper and middle	6	12.2
Left middle and lower	5	10.2
Right middle and lower	2	4.1
Multiple bilateral	1	2.1

it is true that the classification of lung abscesses on the basis of duration of symptoms is at best somewhat inaccurate, the therapy and prognosis of acute and chronic lung abscesses are not the same; most writers, therefore, have attempted to classify their cases strictly as acute or chronic.

Location of Abscess

In this series abscesses of the right lung predominate by a ratio of almost 2 to 1 (table 6). Thirty-two of the 49 cases (65.3 per cent) occurred on the right side, and 17 (34.7 per cent) on the left. This ratio is similar to those reported by D'Ingianni, Woods and McGrath, and Hedblom. The lower lobe of the right lung was involved much more frequently than the upper. Of the 13 cases confined to either the right upper or the right lower lobe, 11 were in the right lower lobe, and 2 in the right upper lobe. Most of the other reports also state that the right lower lobe is the one most frequently involved, although Cutler and Gross[8] found a larger number of abscesses in the upper lobe.

Of greater importance is the fact that multiple lobes of the lungs were involved in 25, or 51 per cent of our 49 cases. This incidence is more than twice that reported by D'Ingianni (25 per cent). Multiple bilateral abscesses were present in 2.1 per cent of our series. This incidence is closely comparable to that reported by Valle (2.5 per cent).

Types of Operations Performed

Operations were performed in 35 of our 49 cases. The procedures employed are listed in table 7. The large number of lobectomies and pneumonectomies was made necessary by the high percentage of chronic abscesses

8. Cutler, E. C. and Gross, R. E.: Nontuberculous Abscess of the Lung; Etiology, Treatment, and Results in 90 Cases. J. Thoracic Surg. 6:125-155 (Dec.) 1936.

Table 7

Types of Operations Perfor.ned and Operative Mortality

Operation	*No. Cases*	*No. Deaths*	*Cause of Death*	*Mortality Rate*
Pneumonectomy	8	5	Air embolus (2) Abscesses in remaining lung (1) Bronchopleural fistula (1) Spread of tuberculosis (1)	62.5%
Lobectomy (2 lobes)	7	3	Air embolus (1) Hemorrhage (1) Bronchopleural fistula (1)	42.6%
Unroofing of abscess	9	3	Hemorrhage (2) Air embolus (1)	33.3%
Lobectomy (single)	7	1	Bronchopneumonia	14.3%
Tube drainage of abscess	2	0		0
Cautery lobectomy	1	0		0
Inter-rib thoracotomy	1	0		0
TOTAL	35	12		34.3%

and multiple lobe involvement. Unroofing of abscesses (carried out in all cases with the cautery, leaving the abscess lightly packed with iodoform gauze), cautery lobectomy, tube drainage, and inter-rib thoracotomy were usually done in acute and "hyperacute" cases[5,9]. Other writers[10] have pointed out that most acute abscesses can be treated by early surgical drainage, but that once the condition of chronic pulmonary suppuration has been reached, cure is rarely possible except by extirpation.

Sweet[10b] listed the following reasons for choosing primary lobectomy for lung abscesses in 15 cases: chronicity of the disease, 10 cases; hemorrhage, 2 cases; involvement of more than one lobe, 1 case; mistaken diagnosis of carcinoma, 1 case; and abscess located in upper lobe, 1 case. Lindskog[11] found primary lung resection the procedure of choice if one or more of the following conditions existed: (1) multiple abscesses or extensive destruction in one or more lobes; (2) secondary bronchiectasis; (3) atelectasis or pneumonitis unrelieved by bronchoscopic treatment; (4) uncontrolled bleeding; and (5) perforation and localized empyema. These writers have done many secondary resections in patients who had persistent disease following drainage of the abscesses. The indications for lobectomy and pneumonectomy in our series were similar to the ones outlined above.

In table 8 the lobectomies are classified according to the lobe removed. One patient had a right lower lobectomy for a large abscess due to tuberculosis. Of the eight pneumonectomies carried out (table 8), 5 were on the right side and 3 on the left. In one of the patients on whom a left pneumonectomy was done, the abscess was found to be a large area of central necrosis in a bronchogenic carcinoma.

Table 8

Portions of Lungs Resected

	No. Cases	*Per Cent*
Lobectomies—Lobes Removed		
Left lower	4	28.6
Right lower	3	21.4
Right middle and lower	2	14.3
Right middle and upper	2	14.3
Left upper	2	14.3
Right upper	1	7.1
TOTAL	14	100
Pneumonectomies—Lung Removed		
Right	5	62.5
Left	3	37.5
TOTAL	8	100

Postoperative Complications

The three most common postoperative complications in this series (table 9) were (1) empyema with broncho-pleuro-cutaneous fistula, (2) bronchopleural fistula, and (3) hemorrhage. Brantigan and Looper[7] reported that broncho-pleuro-cutaneous fistula developed in all but one of 8 cases on which they performed a primary lobectomy for abscess. Iselin[10d] stated that fistula and sec-

9. Neuhof, H. and Touroff, A. S. W.: Acute Putrid Abscess of the Lung: Hyperacute Variety. J. Thoracic Surg. 12: 98-106 (Oct.) 1942.
10. (a) Shaw, R. R.: Pulmonary Abscess; Value of Early, One-Stage Operation. J. Thoracic Surg. 11:453-466 (April) 1942; (b) Sweet, R. H.: An Analysis of the Massachusetts General Hospital Cases of Lung Abscess from 1938 through 1942. Surg., Gynec. & Obst. 80:568-574 (June) 1945; (c) Brown, C. J. O.: Putrid Lung Abscess. M. J. Australia 1:107-109 (Jan. 26) 1946; (d) Iselin, M.: Les abces du poumon d'apres 46 cas operes. Presse med. 54:197-199 (March 30) 1946.
11. Lindskog, G. E.: The Surgical Treatment of Chronic Pulmonary Abscess. Surgery 15:783-788 (May) 1944.

Table 9

Postoperative Complications

Complication	*No. Cases*	*Per Cent*
Empyema, broncho-pleuro-cutaneous fistula	13	54.1
Bronchopleural fistula	5	20.8
Hemorrhage	3	12.5
Bronchopneumonia	1	4.2
Spreading pneumonitis	1	4.2
Dehiscence of wound	1	4.2
TOTAL	24	100

ondary hemorrhage are the most dreaded postoperative complications. Sweet[10b] pointed out that the most frequent cause of death after a drainage operation is spreading infection in the lung.

Mortality

Eighteen of our 49 patients died, giving an overall mortality of 36.7 per cent. Neuhof[5] recently reported the low mortality rate of 2.3 per cent in 172 cases of *acute* putrid lung abscess. Sweet's mortality rate in a series of 120 lung abscesses was 27.3 per cent[10b]. In a series of 2,115 cases of lung abscess collected from the literature, the mortality rate was 34.3 per cent[12]. Freedlander[13] reported a mortality of 40.3 per cent in a series of 238 cases. Overholt and Rumel[14] had a 6 per cent operative mortality in patients with acute lung abscess treated by external drainage. Their cure rate was 94 per cent. In 28 complicated lung abscesses treated by external drainage, they reported an operative mortality of 32 per cent and a cure rate of only 26 per cent. Crimm[6] reported that the mortality in a series of cases receiving bronchoscopic and surgical drainage combined was 16 per cent.

In our series the mortality rate in the 35 patients who were operated upon was 34.3 per cent (table 7). Twenty-three of the patients (65.7 per cent) are now living. Five of the 12 operative deaths followed pneumonectomy, 2 being due to air emboli, 1 to abscesses in the remaining lung, 1 to bronchopleural fistula, and 1 to miliary spread of a tuberculous infection. Three deaths followed the removal of two lobes of the lung, and were caused by air embolus, hemorrhage, and bronchopleural fistula respectively. The mortality from unroofing procedures was 33.3 per cent; 2 of the 3 deaths in this group were due to postoperative hemorrhage, and 1 was due to air embolus. Among the 7 patients on whom simple lobectomy was performed there was only 1 death, and this was due to bronchopneumonia.

In the 14 patients not operated upon, either because they were moribund and expired before operative intervention, or because they refused operation, the mortality rate was 42.9 per cent. Only 8 of the 14 patients are now living.

Results

Of the 31 living patients, 25 (80.6 per cent) have recovered. Four (12.9 per cent) are unimproved, having refused surgical intervention. Broncho-pleuro-cutaneous fistula is still present in 3 (9.7 per cent). It is believed that these patients will probably recover completely, either through spontaneous closure of the fistula or possibly with the aid of further surgery.

In our total series of 49 patients with lung abscesses the recovery rate is 51 per cent. Sweet[10b] found that the total number of cures obtained in the patients treated from 1938 to 1942 was 59.2 per cent. Moore[15] had an apparent cure rate of 55.1 per cent out of 78 patients operated upon; 12.8 per cent have broncho-pleuro-cutaneous fistulas and 6.4 per cent have healed wounds but are still troubled with persistent cough and sputum.

Summary and Conclusion

Forty-nine cases of lung abscesses have been reviewed. In 79.6 per cent the condition had reached the chronic stage before the patient was admitted.

The overall mortality rate in this series was 36.7 per cent, and the mortality rate in the cases operated upon was 34.3 per cent. Sixty-three and three tenths per cent (31 of 49 patients) are living, and 80.6 per cent of the living patients have recovered.

Recent reports in the medical and surgical literature[10c,16] on the use of penicillin by aerosol insufflation, together with intrapleural and intramuscular injections, offer great hope for improvement in the medical treatment of acute lung abscesses *before* pul-

12. Allen, C. I. and Blackman, J. F.: Treatment of Lung Abscess, J. Thoracic Surg. 6:156-172 (Dec.) 1936.
13. Freedlander, S. O.: Total Pneumonectomy, Ohio State M. J. 33:769-776 (July) 1937.
14. Overholt, R. H. and Rumel, W. R.: Factors in the Reduction of Mortality from Pulmonary Abscess, New England J. Med. 224:441-554 (March 13) 1941.
15. Moore, R. L.: Pulmonary Abscess—A Surgical Problem, Ann. Surg. 116:373-386 (Sept.) 1942.
16. (a) Valle, A. R. and White, M. L., Jr.: Penicillin in Pulmonary Resection, J. Thoracic Surg. 14:437-444 (Dec.) 1945; (b) Smyth, C. J. and Billingslea, T. H.: The Treatment of Lung Abscesses with Penicillin; Report of 4 Cases, J.A.M.A. 129:1005-1010 (Dec. 8) 1945.

monary destruction and mixed infection have occurred. In patients requiring surgery for lung abscess, the use of the antibiotics and chemotherapeutic agents now available may help to reduce the morbidity and increase the recovery rate, although little on this subject has appeared in the literature.

Discussion

Dr. Josiah C. Trent (Durham): Dr. Hightower is to be commended for his excellent analysis of the cases of lung abscess treated at the Baptist Hospital. To get some idea of our recent results I have briefly reviewed the 34 cases of lung abscess treated on the thoracic surgical service of Duke Hospital during the year 1946.

All of these cases could be called chronic. Approximately a third were located in the right upper lobe, the left lower lobe being the next most common point of localization. In approximately two thirds of the 34 cases the cause was undetermined. Two cases followed pneumonia, 3 were postoperative, 2 were due to carcinoma, 1 to a cyst, 1 to a foreign body, and 1 to blastomycosis infection.

In 13 of these cases operation was refused or was considered inadvisable. Nine were drained, 9 had lobectomies, and 3 had pneumonectomies. Of those patients who had no operation, 1 died, 7 were discharged unimproved, and 5 improved or well. Of those patients who had drainage, 1 died, and 3 were discharged improved. All the patients who had resections were improved or well when discharged. One patient died of an unknown cause four months after right upper lobe lobectomy. There was one case of postoperative empyema following a pneumonectomy. Most of the patients were treated with penicillin or sulfonamides, or both, and with postural and bronchoscopic drainage.

Chronic lung abscess is a very discouraging disease. However, our entire concept of the treatment of this condition has been radically changed by two factors: (1) penicillin, and (2) refinement of operative technique which permits pulmonary resection with a minimum of risk. The present trend is toward more radical surgery in those cases which do not respond to non-operative therapy, since the danger of acute pneumonic spread and postoperative empyema is minimized by the use of penicillin. Resection offers a chance of cure to many patients who heretofore were condemned to wear a drainage tube with all of its attending miseries, and who were offered little hope of a complete cure.

Knowledge and wisdom—Although there has been a great extension of knowledge in recent years, there has been no accompanying increase in man's powers of reasoning. In fact the reverse seems to have been the case. The physician's mind is so much occupied in the attempt to accumulate more and still more recent knowledge, much of it ill-defined, that his powers of logical reasoning and his sense of perspective are endangered and he is apt to place undue significance and reliance on a specialist or laboratory report, to regard them as being above criticism, and to fail to give due weight to a well-taken history and a careful bedside examination. Knowledge comes, but wisdom lingers, and in no field of human endeavor is this more true than in medicine.—Leslie Hurley: The General Practitioner and the Specialist, M. J. Australia 1:67 (Jan. 17) 1948.

THE MODERN CONCEPTION OF LIVER DISEASES

WILLIAM B. DEWAR, M.D.

RALEIGH

Twenty-five years ago a diagnosis of liver disease or liver dysfunction was frequently made by the family physician; "biliousness" was the word most often used. During my early years of medical practice, terms such as "stimulating the liver," "toning the liver up," and "making the liver act" were much in vogue. The favorite drug for correcting liver abnormalities was calomel in large doses. If one dose did not help, a second dose was immediately given. In those days calomel was given to more patients than sulfadiazine is today—and in my neck of the woods that is saying a lot.

Observing my fellow practitioners giving calomel and getting results, I judged it to be essential that the young doctor follow suit. As a matter of necessity, I became a calomel prescriber. The patient was made quite sick by the large dose of this drug, and of course felt much better when the toxic effects were past. Many patients had to stay at home a day or so while taking the drug, because of the associated disability. Of course, when he saw the green, bile-colored stools, the patient knew that the liver had begun to act and felt that recovery was really on its way. The psychic effect of these stools was marvelous. The people in my community had been taught that they lived in a calomel belt, and as a matter of routine most of them took calomel twice a year to stimulate the liver function. As a liver-toner with calomel I was almost a complete flop, and for a while it appeared that this young doctor would lose caste with both patients and fellow practitioners because of his lack of skill in this art.

During the past ten years the liver dysfunctions have taken their place in the realm of metabolic diseases. The older methods of treatment are being abandoned, and new measures are being adopted which are quite different from those previously in use. We are now having a moderate degree of success in the treatment of diseases of the liver, whereas formerly attempts to treat the more

Read before the Section on the Practice of Medicine, Medical Society of the State of North Carolina, Virginia Beach, Virginia, May 14, 1947.

serious forms of liver dysfunction were considered useless. No definite rules can be put down for the diagnosis and treatment of diseases of this organ, but we can approach the syndrome of liver insufficiency with some degree of hopefulness.

Acute Hepatitis

Chronic liver diseases usually follow acute conditions of the liver. It would seem wise, therefore, to consider the acute processes first. Acute hepatitis arises from damage to this organ by bacterial, chemical, protozoal, or viral agents. The most important, and most prevalent, form of hepatitis is that due to a transmissible filtrable virus. This type is most commonly classified as an infectious hepatitis, and includes the old catarrhal jaundice and many other forms of liver necrosis in which jaundice occurs.

Infectious hepatitis is no new disease. The causal agent has been shown to be present in the blood, nasopharynx, and feces of patients suffering from this disease. It can be spread by infected water and food. The possibility that flies may carry the virus from feces to food must be considered.

The usual incubation period is twenty to forty days, and the onset may be similar to that of many other acute infections. If the disease is sudden in its onset, chills, fever, headache, and aching in the joints and muscles are usually present. The fever is seldom high, and usually disappears after a few days. Blood studies show leukopenia, with relative lymphocytosis; usually no anemia is present, unless the disease is associated with previous liver injury. In about a third of the cases the onset is insidious, and is frequently marked by gastro-intestinal symptoms, including anorexia, nausea, vomiting and diarrhea. Often the patient will complain of abdominal pain, and physical examination will show an enlarged tender liver. If a careful history is taken, the passage of dark-colored urine will often be reported; this color is the result of bile pigments present in the urine. Except in the severe or fatal cases, the patient is usually sickest during the pre-icteric stage.

When the stage of jaundice is reached, all active symptoms may subside, leaving the patient much more comfortable than during the prodromal period. Jaundice usually occurs on the fourth or fifth day after the onset of symptoms, but may be visible the first day, and occasionally comes quite late. It usually persists for two to three weeks, but occasionally lasts months. Itching of the skin is often associated with the jaundice.

Approximately 85 per cent of the patients with viral hepatitis have a spontaneous recovery in forty to fifty days. The disease is usually benign and quite often self-limited. Lucke and Mallory have recently reported a rather large number of cases of epidemic hepatitis which have ended fatally within ten days. Generally speaking, however, the mortality rate is very low.

Diagnosis

The disease is frequently misdiagnosed as influenza, upper respiratory infection, infectious mononucleosis, atypical pneumonia, or obscure fever. Sometimes the erroneous diagnosis is persisted in even after the jaundice has taken place. Differentiation from biliary or hepatic obstruction is very important. Malignant diseases, intercurrent hepatitis associated with cirrhosis, and occasionally hepatitis associated with stones in the gallbladder or ducts are the most important diseases producing such obstruction. The jaundiced patient with acute hepatitis is a poor surgical risk, and if the diagnosis is not positive a reasonable delay should be allowed before operation is attempted. Duodenal drainage, especially if it is combined with urine and stool examinations, may be helpful in diagnosing intrahepatic and obstructive jaundice.

The diagnosis of infectious hepatitis can frequently be made in the pre-icteric stage of the disease. In cases where clinical icterus is not present, one may suspect hepatitis from the history and from the finding of a large, tender liver on physical examination. In certain cases liver function tests are very helpful in diagnosis. The tests which are most reliable are the bromsulfalein retention test, determination of plasma bilirubin, the thymol turbidity test, and the cephalin-cholesterol flocculation test. The Harrison spot test for bilirubin in the urine may be most valuable at times. In applying these tests, one must remember that 80 per cent of the liver may be destroyed without impairment of its function. The liver's capacity for regeneration is astounding, and a large quantitative loss of the hepatic parenchyma may be replaced in a short period of time.

Many physicians believe that the nonicteric form of hepatitis occurs more frequently than the icteric form, and often goes undiagnosed. It is suspected that this form of hepatitis may be the forerunner of subsequent chronic liver insufficiencies and hepatic cirrhosis. The liver function tests usually show more abnormality in the early stages of the disease.

Jaundice has been recognized with increasing frequency following transfusions of plasma, whole blood, and serum. This type of infectious hepatitis usually occurs sixty to one hundred days after the injection. Danger of producing this type of hepatitis is always present when patients are given blood, plasma, or serum. No person who has been jaundiced in the past twelve months should be used as a donor. Stokes of Philadelphia has presented information that would make us believe that 10 cc. of gamma globulin given intramuscularly may prevent acute hepatitis.

Treatment

There is no specific treatment for acute hepatitis. In the active stage of the disease gamma globulin given intramuscularly is without effect. Bed rest and diet are the two most important factors in treatment. Delay in instituting bed rest will lengthen the course of the disease, and if the patient overexerts too soon, he may have a relapse. A diet high in carbohydrates and protein, and reasonably high in fat should be given. The usual requirement is 300 Gm. of carbohydrates, 125 of proteins, and at least 100 of fat daily. Recently Hoagland has given patients with infectious hepatitis a high-fat diet, and believes that it may be helpful rather than harmful, as was originally reported. The fat should usually be limited to butter, milk, eggs, and that present in boiled or baked meat.

Patients with hepatitis often have severe anorexia and nausea. In these cases it is difficult to maintain a proper nitrogen balance, especially in the early stages of the disease. At times glucose and protein hydrolysates may be given intravenously with excellent results. If parenteral feedings are indicated, vitamin supplements should be added. In patients with severe vomiting, plasma and whole blood are helpful.

Intramuscular injections of vitamin K are indicated if the prothrombin time of the blood is delayed. Sulfadiazine and streptomycin should not be used unless urgently needed to combat a complicating bacterial infection. Penicillin in large doses every two hours may occasionally be life saving if the hepatitis is bacterial in origin.

Patients with liver disease tolerate barbiturates and opiates poorly. Alcohol is absolutely contraindicated, as it is in all forms of liver insufficiency. Most authorities believe that it should not be used for at least six months after the patient has made a complete recovery.

A few patients are very slow in recovering from viral hepatitis, and have a prolonged convalescence. Successful management of these patients requires skill and ingenuity. Frequently they do not appear ill, but complain of weakness, fatigue, and general malaise. They may be classed as neurotic, unless their condition is properly evaluated. The capacity of the patient to accomplish physical exercise is the best measure of convalescence. Middleton has called attention to the importance of graded exercise. Exercise tolerance tests may be helpful when graduated properly after infectious hepatitis. No patient should be classed as cured until he can take real exercise—that is, walk long distances without adverse symptoms.

Chronic Hepatic Insufficiency

In a small number of cases, acute hepatitis may develop into chronic liver disease. Early recognition and proper treatment of the acute condition will prevent this development in a large percentage of the cases. In chronic hepatitis, jaundice may come and go. This type of jaundice is frequently seen in heavy drinkers with hepatic insufficiency, which may progress over a period of months or years to the stage of real cirrhosis. On the other hand, symptoms may abate in the late stage of chronic hepatitis so that they no longer interfere with the patient's activity.

Frequently hyperbilirubinemia and marked hypoproteinemia may be present in chronic liver disease, and may be accompanied by ascites and edema. The findings in cirrhosis resulting from chronic hepatitis are not unlike those in cirrhosis arising from other causes. For all practical purposes Laennec's cirrhosis and chronic liver

insufficiency may be discussed together. The most important factor in the treatment of these diseases is their prevention whenever possible. Anorexia, loss of weight, anemia, dye retention in the liver, and reversal of the albumin-globulin ratio are signs of chronic liver disease which appear long before real cirrhosis with edema and ascites develops.

Etiologic factors

Snell and others have called attention to the fact that the nutritional disturbances associated with heavy drinking, rather than the toxic effect of alcohol on the liver, may be the cause of cirrhosis. Cirrhosis has been found in people who use no alcohol, but whose dietary intake is markedly deficient in proteins and vitamins. Among heavy drinkers real cirrhosis of the liver occurs less frequently in those who eat an adequate diet.

Treatment

Patients with chronic liver disease should be given the same diet used in acute infectious hepatitis. It is frequently difficult to maintain an adequate caloric intake in these patients, because of gastrointestinal complaints and anorexia. Concentrated and frequent feedings may be helpful. If they cannot be taken, intravenous injections of glucose and protein hydrolysates should be employed when all other efforts to provide an adequate caloric intake fail.

In order to obtain results with crude liver extract, it is believed that larger doses are required than can be given intramuscularly. A recent issue of the *Journal of the American Medical Association* carries a report on the use of liver extract intravenously in cirrhosis of the liver[1]. Apparently excellent results have been obtained in some cases by this method. The authors report an increase in the patient's appetite and sense of well-being, along with disappearance of ascites in some cases.

We have had very little experience with the intravenous use of liver extract in treating cirrhosis of the liver. Intraheptol[2] is a preparation that we have used a few times. Careful tests for sensitivity must be carried out before administration of the full intravenous dose is attempted. A small dose is first given intramuscularly. If no reaction occurs, 1/20 cc. of the liver extract diluted with 1 cc. of normal salt solution may be given slowly by vein. This injection should take at least three to five minutes. After thirty minutes, if no untoward effects are noted, 1 cc. of the liver extract diluted with 9 cc. of normal salt solution may be given very slowly by vein. If no reaction occurs, 2 cc. may be given the following day, with 8 cc. of saline solution. The dose is gradually increased until 10 cc. of crude liver is given with 40 cc. of normal saline solution. Each injection should require at least five to eight minutes. It is recommended that 10 cc. be given intravenously three times a week for several months.

We now know that many of the symptoms of chronic liver disease arise from the fact that the organ cannot properly metabolize protein. Quantitative and qualitative changes in plasma protein frequently occur, and may cause ascites and edema. In such cases intravenous use of human albumin may produce remarkable clinical improvement. Edema may be relieved in a few days, and even ascites may decrease. Fifty to 100 Gm. may be given at weekly intervals until a satisfactory plasma level of albumin is obtained. The patient should be kept in positive nitrogen balance. We have had no experience with the use of homologous albumin. It is recommended that when albumin is not available 500 to 750 cc. of plasma be given two or three times a week. Certainly, plasma has been helpful in many cases. Methionine and choline may be of some benefit in chronic liver disease. We have had no experience with these substances, but it would seem from the evidence at hand that in patients who can take an adequate diet little benefit can be expected from their use.

Chronic liver disease takes on eventually the characteristics of multiple vitamin deficiency. The damaged liver is unable to carry on adequately the metabolism of numerous constituents essential for maintenance of the body economy. Any measure helpful in overcoming the metabolic defect should be included in the therapy. When the measures outlined above are used early enough, they may be effective in at least checking liver disease, and occasionally the pathologic processes are reversed.

1. Labby, D. H., Shank, R. E., Kunkel, H. G., and Hoagland, C. L.: Intravenous Therapy of Cirrhosis of the Liver, J.A.M.A. 133:1181-1190 (April 19) 1947.
2. Put out by Lederle Laboratories.

Conclusion

In the diagnosis and treatment of liver disease, experience, common sense, and good judgment are essential for the best results.

Discussion

Dr. George T. Harrell (Winston-Salem): At the Bowman Gray School of Medicine we have actually made determinations of the vitamin levels in cirrhosis. Cayer in our clinic, studying the results of therapy in cirrhosis of the liver, has found that the large doses of some vitamins commonly used may actually be harmful (Arch. Int. Med. 80:644-654 (Nov.) 1947). He has shown by a study of the urinary excretion of F_2, a fluorescent substance, that the damaged liver cannot utilize and break down niacinamide. We now prefer to use a daily vitamin supplement containing 10 to 15 mg. of niacinamide rather than the usually recommended dose of 100 to 150 mg.

It has been our impression that a high-protein, high-vitamin diet, supplemented with sulfur-containing amino acids, not only has a lipotropic action but definitely aids in the regeneration of liver cells. We believe that a full and adequate diet is equally as effective as the oral or intravenous administration of amino acids. If the patient is unable to eat the diet, we have forced feedings through an indwelling duodenal tube. Dramatic responses have been obtained by such techniques in patients who were comatose and appeared to be hopelessly ill. In one instance the liver shrank so rapidly from day to day that we were afraid we had induced acute necrosis; the subsequent complete recovery of the patient, however, indicated that the reduction in size was due to the mobilization of fat at an extremely rapid rate.

Lipotropic substances have been found to be of little value in the therapy of acute virus hepatitis.

Cancer mimicking ulcer . . . is seen by every surgeon several times a year. It is necessary to remind ourselves that cancer of the stomach, while it is usually a very rapid disease, may be as slow as a scirrhous tumour of the breast, going on for ten years with little change; that patients with slowly progressing cancers may have a good appetite and free hydrochloric acid, and that they may lose their symptoms for a time and put on weight on a gastric diet. These slow cancers are digested in their central necrotic parts by the gastric juices, and, whether at operation or necropsy, may have very much the appearance of an indurated peptic ulcer. But in these cases, if we go into the history carefully, we find that the gastric symptoms have started suspiciously late in life, that though they may have gone for some years they have been steadily and slowly progressive, and that the remissions have not been periods of complete health but of lessened discomfort. The true ulcer usually starts in the twenties, and, in its long history before its final cure by medicine or surgery, has many remissions of months' or years' duration. I know that prolonged irritations and the presence of scar tissue may give rise to a cancer, but in a lifetime I have seen little evidence that they do so except in squamous epithelium.—Ogilvie, H.: The Early Diagnosis of Cancer of the Oesophagus and Stomach, British M. J. 2:407 (Sept. 13) 1947.

One in every 25 World War II veterans training under the G. I. Bill is preparing for a career in medicine or in a related field, a Veterans Administration survey revealed.

CHRONIC GLOMERULONEPHRITIS WITHOUT HYPERTENSION, CARDIAC ENLARGEMENT, OR RETINAL CHANGES

Case Report with Autopsy Findings

CLIFFORD G. GADDY, M.D.
and
DAVID CAYER, M.D.
WINSTON-SALEM

Uremia is a syndrome resulting from renal insufficiency and accompanied by retention of urinary constituents. It is characterized by signs and symptoms related to the circulatory, gastrointestinal, respiratory, dermatologic, and nervous systems. Most varieties of renal disease which lead to uremia are associated with hypertension and hypertensive changes. Amyloid disease of the kidney and necrosis of the tubules due to mercury are exceptions, and even these conditions may produce hypertension in the late stages.

Probably the most common cause of uremia is chronic glomerulonephritis. The diagnosis of this condition, as a rule, presents no difficulty. In typical cases, the hypertension, cardiac enlargement, retinitis, albuminuria, and hematuria constitute an unequivocal picture. In patients with severe chronic glomerulonephritis, the blood pressure may drop because of cardiac weakness, but the presence of cardiac enlargement gives evidence of the antecedent hypertension. When the constant features are absent, however, the disease may be more difficult to diagnose.

Instances of adults dying in uremia due to chronic glomerulonephritis without hypertension or cardiac hypertrophy are rare. Fishberg[1] and Bertola[2a] each observed a single case, and Bannick[2b], Foster[2c], and Weissberg and Ippolito[2d] each reported 2 cases of chronic glomerulonephritis without

From the Department of Medicine, Bowman Gray School of Medicine of Wake Forest College and the North Carolina Baptist Hospital, Winston-Salem, North Carolina.

1. Fishberg, A. M.: Hypertension and Nephritis, ed. 4, Philadelphia, Lea & Febiger, 1939, p. 170.
2. (a) Bertola, A.: Contributo allo studio delle nefriti croniche iperazotemiche anipertoniche, Gazz. d. osp. 60: 967-974 (Oct. 22) 1939; (b) Bannick, E. G.: Severe Chronic Glomerular Nephritis without Hypertension, Cardiac Hypertrophy or Retinal Changes; Report of Two Cases, Arch. Int. Med. 39:741-747 (May) 1927; (c) Foster, N. B.: The Relations of Hypertension to Cardiorenal Diseases, Am. J. M. Sc. 164:808-819 (Dec.) 1922; (d) Weissberg, J. and Ippolito, T.: Diffuse Glomerulonephritis without Hypertension, Bull New York M. Coll., Flower & Fifth Ave. Hosps. 3:12-21 (June) 1940.

hypertension or cardiac enlargement. Several other reports of similar cases have appeared in the literature, but many were not confirmed by postmortem observations.

It is felt that the rarity of apparently uncomplicated renal failure due to chronic glomerulonephritis justifies report of the following case.

Case Report

A white automobile mechanic, aged 48, entered the North Carolina Baptist Hospital on January 15, 1947, complaining of headaches, weakness, anemia, and a generalized eruption. He also had numbness in the lower extremities, anorexia, a mild postnasal drip, and occasional nocturia. He had been in good health until a year before admission, when he began having moderate weakness and slight dyspnea. Six months prior to admission he acquired a rash which involved the entire body except the face. The lesions were described as being papular, erythematous, and pruritic. A month later he was told that he was anemic, and was treated with intramuscular liver extract and iron. Three months of this therapy produced no improvement. Two and a half months prior to admission his hemoglobin was 50 per cent.

Two weeks before he was seen in this hospital, examination at another clinic revealed a generalized maculopapular rash which did not involve the face, a peculiar yellow, bronzed discoloration of the skin, and a left inguinal hernia. A blood count at that time showed 6.2 Gm. (40 per cent) of hemoglobin and 1,830,000 red blood cells. The red cells showed slight anisocytosis, poikilocytosis, and basophilic stippling; an occasional macrocyte was seen. The reticulocyte count was 10 per cent. Gastric analysis showed no free hydrochloric acid after histamine.

The patient stated that he had consumed an average of one quart of liquor a week for many years, but none for the past seven years. He had had occasional episodes of epistaxis for several years, but since the present illness began nosebleeds had been frequent and at times severe. He had occasional colicky pain, nausea, and vomiting. His appetite had been poor for the past two months, and he had lost 30 pounds during this time.

Physical examination showed the temperature to be 98.6 F., pulse 116, respiration 22, blood pressure 138 systolic, 84 diastolic. The patient was a well developed, well nourished, white man with pallor and slight dyspnea. There was a maculopapular eruption over the arms, legs, and body, with excoriation, crusting, and bleeding scratch marks. The pupils and the optic fundi were normal. Clotted blood was present in the left nostril. The lungs were clear throughout and the heart was not enlarged. The rhythm was regular and no murmurs were heard. The liver was palpable three fingers' breadth below the right costal margin. A reducible left inguinal hernia was present. There was no edema of the extremities and neurologic examination was essentially normal.

Urinalysis showed a specific gravity of 1.008, and a 1-plus reaction for albumin; on microscopic examination occasional red and white blood cells were seen per high power field. The hemoglobin was 7.3 Gm., red blood cells 2,150,000, reticulocytes 1.8 per cent. The urine contained no Bence-Jones protein and the Sulkowitz test was negative. The total serum proteins were 6.3 Gm. per 100 cc., with 4.3 Gm. of albumin and 2.0 Gm. of globulin. Serologic tests for syphilis were negative. Examination of the stool was negative for occult blood. A gastric analysis (fasting specimen) showed 24 clinical units of free hydrochloric acid and 50 of combined acid. Examination of the bone marrow showed depression of the erythroblastic series. Fluoroscopy of the chest, an x-ray of the skull, and an abdominal series were negative.

The patient's course in the hospital was rapidly down hill. On January 17 a furuncle appeared on the left thigh. The temperature ranged from 98 to 101 F.; the blood pressure remained at 140 systolic, 80 diastolic. Increasing dyspnea and signs of marked acidosis developed. The nonprotein nitrogen was found to be 164 mg. per 100 cc. of blood, and the carbon dioxide combining power 5 volumes per cent. The dyspnea was relieved by sixth molar lactate given intravenously. Repeated phenolsulfonphthalein tests showed no excretion of dye. The blood nonprotein nitrogen increased to 250 mg. per 100 cc. On January 22, generalized tetany developed, and the patient became cyanotic. Calcium gluconate and aminophyllin produced immediate improvement. The blood pressure was 210 systolic, 110 diastolic following tetany, but afterwards fell again to normal. On January 22 respirations became deep and irregular, with frequent apnea. The heart sounds became faint, and the patient expired.

Autopsy findings

The anatomic diagnoses were: (1) generalized excoriation of the skin, (2) abscesses of the left hip, (3) generalized arteriosclerosis, (4) hemangioma of the liver, (5) mitral valvulitis, healed, and (6) chronic glomerulonephritis.

The kidneys were equal in size, and together weighed 145 Gm. They were pale red, and the surfaces showed many small cysts and petechiae. The capsules were easily stripped, leaving a coarsely granular surface. A cross section showed bilateral cysts. The diameter of the renal cortices measured 4 mm. on the right and 3 mm. on the left; the diameter of the pyramids was 10 mm. on the right and 8 mm. on the left. The pelves and ureters were normal.

Microscopic examination showed decrease in the number of glomeruli and marked variation in their size. Many were hyalinized and completely fibrosed, while those remaining showed epithelial hyperplasia and many healed crescents. The tubules were decreased in number, and many were distended. There were many cystic spaces of varying size, lined by a single layer of flattened epithelium. Infiltration of the interstitial tissues by small round cells was noted. The arteries showed internal thickening, with vacuolation and hyalinization of the media. No arteriolar changes were noted.

The heart weighed 375 Gm. The left ventricular wall measured 1.5 cm. in diameter. A few nodular thickenings about 2 mm. in diameter were seen along the valve edges. There was no stenosis or thickening of the valve margins.

The liver was slightly enlarged and showed moderate central necrosis.

Differential Diagnosis

The absence of hypertension, retinitis, physical signs of cardiac enlargement, and significant urinary findings or a history of previous urinary complaints in this patient obscured the true diagnosis. Carcinoma of the stomach was suggested because of the weakness, anemia, weight loss, nausea, and vomiting. Other writers[2] have found gas-

trointestinal malignancy to be the principal problem in the differential diagnosis of these atypical cases of glomerulonephritis. Other diagnoses considered in this case included Addison's disease, the primary anemias, multiple myeloma, and aplastic anemia.

Possible Explanations for the Absence of Hypertension

The modern concepts of renal hypertension hold that the ischemic kidney liberates a proteolytic enzyme, *renin*, which acts on a pseudoglobulin, *hypertensinogen* or *angiotonin precursor*, to split off a pressor substance, *hypertensin* or *angiotonin*, which is probably a polypeptide. Hypertensin is destroyed both *in vivo* and *in vitro* by the action of an inhibitor, *hypertensinase* or *angiotonase*, present in the blood and in various organs. There is evidence that renin itself is antigenic and may cause formation of *antirenin*. The following possibilities might permit the development of renal excretory failure without the production of hypertension.

1. *The absence of renin*

Renin is derived from the kidney cortex. It is apparently produced or stored by the proximal convoluted tubules. Kidneys in which the proximal tubules are poisoned by tartrate do not contain renin[3]. It has been recognized for several years that renal ischemia in animals increases the liberation of renin. Recently, Dexter and his associates[4] have shown that in human beings, as in animals, constriction of the renal artery stimulates the renal humoral mechanism. The conditions essential to the production of renin are not known. There is no good evidence that anoxemia, histotoxic anoxia, or reduced pulse pressure is responsible.

According to Bell[5], experimental hypertension of the Goldblatt type in dogs disappears after the kidney becomes markedly atrophic. Apparently kidneys that have undergone marked atrophy may cease to produce a pressor substance.

2. *The absence of hypertensinogen*

Renin tachyphylaxis, according to Page and his associates[6], may be attributed to the exhaustion of hypertensinogen. This finding seems to indicate that the reserve supply of hypertensinogen is limited. The liver is the chief source of hypertensinogen; in normal and hypertensive dogs removal of the liver, or hepatic damage, reduces the hypertensinogen content of plasma, and hence the pressor response to injected renin[7].

Haynes and Dexter[8] studied the hypertensinogen levels in patients with liver damage, and showed that 6 of 10 patients had lowered values. The most marked decrease was seen in cases of advanced cirrhosis with severe hepatic insufficiency.

3. *The destruction or inactivation of hypertensin by hypertensinase or other proteolytic ferments*

Hypertensin is destroyed by hypertensinase, an albumin fraction found in the blood and in various organs, especially the kidneys[9]. *In vitro* studies have shown that hypertensin is also destroyed or inactivated by trypsin, pepsin, carboxypeptidase, aminopeptidase, extracts of liver and spleen, and fresh normal blood serum[10].

4. *The absence of essential endocrine factors*

In adrenalectomized animals the hypertensinogen levels are lowered and the sensitivity to hypertensin is apparently decreased, while the serum content of hypertensinase is unaltered. Removal of the hypophysis dampens but does not abolish the effects of constriction of the renal arteries[11]. The thyroid, parathyroids, and pineal glands play no significant role in renal hypertension.

Page[12] has shown that only cortin is necessary to maintain hypertension in dogs

3. Best, C. H. and Taylor, N. B.: Physiological Basis of Medical Practice, ed. 4, Baltimore, The Williams and Wilkins Company, 1945, p. 132.
4. Quinby, W. C., Dexter, L., Sandmeyer, J. A., and Haynes, F. W.: Renal Humoral Pressor Mechanism in Man: Effect of Transitory Complete Constriction of the Human Renal Artery on Blood Pressure and on Concentration of Renin, Hypertensinogen and Hypertensinase of Renal Arterial and Venous Blood, with Animal Observations, J. Clin. Invest. 24:69-74 (Jan.) 1945.
5. Bell, E. T.: Renal Diseases, Philadelphia, Lea & Febiger, 1946, p. 310.
6. Page, I. H., and others: Reduction of Arterial Blood Pressure of Hypertensive Patients and Animals with Extracts of Kidneys, J. Exper. Med. 73:7-41 (Jan.) 1941.
7. Page, I. H., McSwain, B., Knapp, G. M., and Andrus, W. De W.: The Origin of Renin-Activator, Am. J. Physiol. 135:214-222 (Dec.) 1941.
8. Haynes, F. W. and Dexter, L.: The Renal Humoral Pressor Mechanism in Man; Hypertensinogen Content of Plasma of Normal Patients and Patients with Various Diseases, J. Clin. Invest. 24:78-81 (Jan.) 1945.
9. Braun-Menendez, E., Fasciolo, J. C., Leloir, L. F., and Munoz, J. M.: The Substance Causing Renal Hypertension, J. Physiol. 98:283-298 (July 24) 1940.
10. Croxatto, R. and Croxatto, H.: Comparative Study of Hypertensinase and Proteinase Activity of Blood Plasma, Proc. Soc. Exper. Biol. & Med. 61:330-333 (April) 1946.
11. Page, I. H. and Sweet, J. E.: The Effect of Hypophysectomy on Arterial Blood Pressure of Dogs with Experimental Hypertension, Am. J. Physiol. 120:238-245 (Oct.) 1937.
12. Page, I. H.: The Effect of Bilateral Adrenalectomy on Arterial Blood Pressure of Dogs with Experimental Hypertension, Am. J. Physiol. 122:352-358 (May) 1938.

with both renal arteries constricted, and with the hypophysis, testes, and adrenals removed.

5. *Inactivation of pressor amines by aminases*

Bing and Zucker[13] have demonstrated that the decarboxilation of 1-dihydroxyphenylalanine by the ischemic kidney produces hydroxytyramine, a pressor substance. Certain amino acids, after decarboxilation without deaminization, yield powerful pressor amines. The demonstration of Schroeder and Adams[14] that tyrosinase (a phenolic oxidase) as well as aminoxidase, prepared from hog liver, lowers the blood pressure in dogs lends support to this possible mechanism of hypertension.

Morton and Tainter[15], studying the effects of several sympathomimetic amines on perfused blood vessels, learned that certain of these amines failed to show activity except in the presence of adrenaline. It appears that these amines act by blocking the ferment which destroys the adrenaline constantly liberated by the blood at the myoneural junction. Such a ferment (the analogue of cholinesterase) has not yet been demonstrated, but is being generally assumed. Absence of adrenaline at the myoneural junction or increased activity of the ferment which destroys adrenaline would inactivate pressor amines formed in the ischemic kidney. Such a disorder, the physiologic analogue of myasthenia gravis, has not been described.

6. *Immunity to renin, hypertensin, or hypertensinogen*

Recent experiments[16] have shown that renal lesions which closely simulate those of human glomerulonephritis can be produced by means of antikidney serums. It is difficult to prove that glomerulonephritis in human beings may be due to specific antibodies acting against renal tissue, since in all cases of experimental glomerulonephritis the antibodies were developed in an animal species other than the one in which the renal lesion was produced. The evidence for auto-antibodies to kidney substance lies in the demonstration that injections of a mixture of kidney substance and streptococcus toxin caused animals to produce antibodies which would precipitate both kidney extract and streptococcus toxin. The production of these antibodies is most readily explained by the concept of haptens—the streptococcal toxin acting as a hapten to render the kidney extract antigenic. That a similar mechanism might lead to the formation of antirenin, antihypertensin, or antihypertensinogen seems possible.

Goldblatt and his associates[17] have shown that when dogs are given small intramuscular injections of hog renin for three to five months a factor develops in the blood which prevents the pressor effect of large doses of hog renin given intravenously. The blood serum of such dogs is also capable of inactivating or destroying renin *in vitro*, so that a mixture of renin and antiserum does not produce a pressor response when injected into other dogs. Wakerlin[18] has concluded that the antihypertensin effect produced by repeated injections of hog renin extract is due to some constituent other than renin. It is possible that some other type of immune response to renin is involved. The possibility of the production of auto-antibodies to renin deserves consideration in this case.

Application of Theories to the Present Case

That some component of the renin-hypertensive mechanism failed to be present, or to exert its usual action, in the case under discussion must be considered. The characteristic microscopic picture of chronic glomerulonephritis makes the existence of renal ischemia certain.

1. In view of our limited knowledge concerning the liberation of renin, it is difficult to presume its absence in this patient. One might speculate that the renin-producing cells—presumably the proximal convoluted tubules— suffered greater destruction, proportionately, than did those producing the antipressor substance (hypertensinase). The autopsy findings do not support this theory, however.

13. Bing, R. J. and Zucker, M. B.: Renal Hypertension Produced by Amino Acid, J. Exper. Med. 74:235-246 (Sept.) 1941.
14. (a) Schroeder, H. A. and Adams, M. H.: Effect of Tyrosinase on Experimental Hypertension, J. Exper. Med. 73: 531-550 (April) 1941; (b) Schroeder, H. A.: Effect of Preparation of Amine Oxidase on Experimental Hypertension, Science 95:306-307 (March 20) 1942.
15. Morton, M. C. and Tainter, M. L.: Effects of Sympathomimetic Amines on Perfused Blood Vessels, J. Physiol. 98:263-282 (July 24) 1940.
16. Cavelti, P. A. and Cavelti, E. S.: Studies on the Pathogenesis of Glomerulonephritis; Production of Autoantibodies to Kidney in Experimental Animals, Arch. Path. 39:148-152 (March) 1945.
17. Goldblatt, H., and others: The Nature and Properties of Antirenin, J.A.M.A. 122:135-136 (May 8) 1943.
18. Wakerlin, G. E., in discussion of Goldblatt (17).

2. Lack or inactivation of hypertensinogen is unlikely. The degree of liver damage found would not appear great enough to justify this explanation.

3. Proof that there was destruction or inactivation of hypertensin can not be given, and the work of Haynes and Dexter[8] makes this possibility unlikely. In a study of patients with hypertension, azotemia, and Addison's or hepatic disease, they concluded that hypertensinase played no significant role in preventing the production of hypertension.

4. Addison's disease was considered clinically in the present case, but no evidence of adrenal destruction was found at necropsy.

5. We have no data to support the theory that inactivation of pressor amines was the mechanism which prevented the development of hypertension in this patient.

6. The formation of antirenin offers a possible explanation for the absence of hypertension in this case. Substantial evidence that auto-antibodies may develop to kidney extract has been presented. That some foreign substance such as a component of crude liver extract, which the patient received, or some bacterial toxin may have served to render the renin antigenic in this patient seems possible. Further studies on the antigenic properties of renin may be revealing.

Summary

A case of terminal uremia due to chronic glomerulonephritis, without clinical evidence of hypertension, cardiac enlargement or retinal changes, and with minimal urinary findings, is reported.

The problem of differential diagnosis and the mechanisms by which renal excretory failure may develop without the production of hypertension are discussed.

The man doing general practice is largely looked up to in his community as a leader. He should not try to shirk such responsibility. He should keep reasonably abreast of local and other politics. Of course it is not good for a doctor to get deeply involved in politics, but he should be able to give good advice at all times. He should keep abreast of all civic matters, particularly where medical questions are concerned. He should be prepared to address local organizations on current matters without being naive. He should make it his business to be acquainted with at least some of his city and county officials so that he can use his influence at the proper time.—E. A. Royston: The Section on General Practice: Its Problems, Its Goals, Its Responsibilities, California Med. 68:281 (April) 1948.

MUCOCELE OF VERMIFORM APPENDIX

With Report of Three Cases

W. B. McCUTCHEON, M.D.
and
RAIFORD D. BAXLEY, M.D.
DURHAM

A true cyst of the appendix containing mucus is known as a mucocele. Actually, the encysted material is pseudomucin, which can be identified by Hammarsten's method as described by Lewis[1], after any protein or mucin has been precipitated and filtered.

Weaver[2] credits Rokitansky with having recognized the condition in 1842, but other authors state that Virchow first described it in 1863. Fraenkel in 1901 reported the first autopsy performed in a case of pseudomyxoma peritonei, in which a ruptured appendix was found containing the same gelatinous substance as the general peritoneal cavity.

Incidence

Dannreuther[3] states that in a series of 8,457 appendectomies at the New York Post-Graduate Hospital, mucocele was found eight times—an incidence of 0.1 per cent. Seven of these cases were in women past middle life. Other authors vary in their reports as to incidence, the figures ranging between 0.3 per cent and 0.11 per cent. Grodinsky and Rubnitz[4] quote Jones and Carmody as stating that, up to 1936, the total number of reported cases of mucocele of the appendix was approximately 400. The incidence of "pseudomyxoma peritonei" is much less, only 90 cases having been reported up to 1934[5].

Etiology

Phemister[6] attributed the cause of mucocele formation to normal appendiceal invo-

From the Department of Surgery, Watts Hospital, Durham, North Carolina.

1. Lewis, E. G.: Pseudomyxoma of the Peritoneum. Surg., Gynec. & Obst. 19:757-760, 1914.
2. Weaver, C. H.: Mucocele of Appendix with Pseudomucinous Degeneration. Am. J. Surg. 36:523-526 (May) 1937.
3. Dannreuther, W. T.: Mucocele of Vermiform Appendix. Am. J. Obst. & Gynec. 31:342-351 (Feb.) 1936.
4. Grodinsky, M. and Rubnitz, A. S.: Mucocele of Appendix and Pseudomyxoma Peritonei. Surg. Gynec. & Obst. 73: 345-354 (Sept.) 1941.
5. D'Aunoy, R. and Fine, A.: Pseudomyxoma Peritonei of Appendiceal Origin. Am. J. Cancer 22:59-65 (Sept.) 1934.
6. Phemister, D. B.: Pseudomucinous Cyst of the Appendix. J.A.M.A. 64:1834-1836, 1915.

lution and associated inflammation. Milliken and Poindexter[7] described the conditions which they believed to be essential to mucocele formation as follows:

(1) A slowly stenosing process must take place, as a rapid stenosis would lead to gangrene.

(2) The lumen must be sterile, as the presence of bacteria would lead to empyema.

(3) An actively secreting mucosa must be present, or secretion must be more rapid than absorption, and there must be a change in the mucosa, so that mucus is changed into pseudomucin.

Waugh and Findley[8] distinguish between *hydrops* and *mucocele*. In the former condition an accumulation of viscid fluid in the lumen of the appendix stretches the mucosa and leads to atrophy and a thinning of the muscular wall; the latter is characterized by retention of thick, viscid mucus, by a hyperplastic mucosa, and by thickened, fibrous, vascularized walls.

McCrae and Coplin[9] offered the theory that mucocele formation is due to a localized area of obstruction near the appendiceal base, caused by inflammation and fibrosis. If the inflammation is extensive, the succeeding fibrosis will obliterate the lumen. Localized fibrosis, however, constricts the lumen and prevents drainage, so that the organ is gradually distended with mucus. The accumulated mucus is changed into a jelly-like material as a result of absorption.

Wells[10] produced an appendiceal mucocele in a rabbit by placing a ligature at the root of the appendix, not including the appendicular vessels. Grodinsky and his coworkers[4] produced mucoceles in rabbits by the same technique. In the animals that developed mucoceles, secondary intraperitoneal deposits were noted at future operations performed after an adequate time interval.

Diagnosis

Mucocele presents no definite symptom complex. One of our patients had a palpable mass in the right lower quadrant; the second patient had symptoms typical of acute appendicitis; and the third case was symptomless. Most of the articles on the subject mention pain as a symptom, but this pain is vague and is not apt to be localized. It may be felt in the epigastrium, para-umbilical region, or right lower quadrant. Fever, leukocytosis, nausea, and vomiting are usually not noted except in the acute superimposed attack. There may be tenderness with or without rigidity over the appendix. Vorhaus[11] reported a case diagnosed preoperatively by barium enema examination, and gave the following criteria to support such a diagnosis:

1. An irregular filling defect of the lower end of the cecum.
2. A large, irregularly filled appendix.
3. A large appendiceal shadow easily confused with the terminal ilium.
4. A cecal defect, showing a series of superimposed striations, as though the barium were filtering through different levels of semiviscid substance.
5. On partial evacuation, a feathered appearance of the lower cecum and appendix.

Usually the obstruction responsible for the mucocele does not allow filling of the appendix. Filling can occur only if the obstruction is incomplete or if the lumen has reopened.

Complications

Carcinoma has been known to develop in a mucocele, and it has been suggested that malignancy might be the obstructive factor producing a mucocele. Intestinal obstruction due to the pressure of the mucocele, or to intussusception, may develop.

The major complication of mucocele is rupture and general peritoneal contamination with the pseudomucinous material. Weaver[2] states that, as the appendix distends, diverticula form. These may rupture and extrude material into the peritoneal cavity, or spillage may be due to rupture of the organ. The appendix may then collapse, the rupture may heal, and the whole process may repeat itself. The extruded material is not absorbed, but becomes encapsulated by the omentum, or by fibrous tissue produced by a local plastic peritonitis. In time, the abdomen becomes distended with gelatinous masses. It is hardly possible that the quantities of material which have been noted in the peritoneal cavity could be extruded

7. Milliken, G. and Poindexter, C. A.: Mucocele of Appendix with Globoid Body Formation. Am. J. Path. 1:397-402 (July) 1925.
8. Waugh, T. R. and Findley, D.: Mucocele with Peritoneal Transplantation in Adenocarcinoma of Appendix. Am. J. Surg. 37:518-525 (Sept.) 1937.
9. McCrae, T. and Coplin, W. M. L.: Gelatinoid Carcinoma (Morbus Gelatinosus) of the Peritoneum. Am. J. M. Sc. 151:475-491, 1916.
10. Wells, A. Q.: Experimental Lesions of Rabbit's Appendix. Brit. J. Surg. 24:766-772 (April) 1937.
11. Vorhaus, M. G.: Recognition of Some Less Common Diseases: Duodenal-Jejunal Diverticula; Mucocele of Appendix and Cecum. J.A.M.A. 94:165-169 (Jan. 18) 1930.

through small openings. It is more probable that the initial seedlings which are extruded proliferate to form a true intraperitoneal adenocystoma.

It is to be emphasized that "pseudomyxoma peritonei" is not an entity in itself. The same picture can be produced by a ruptured ovarian cystadenoma, or the condition may originate in mesenteric cysts, mucoceles, retroperitoneal cystadenomas, or intestinal diverticula. In one reported case[9], it followed cancer of the gallbladder.

Pathology

There is considerable variation in the size of mucoceles. The shape—whether globular or sausage-like—seems to depend on the site of obstruction in relation to the appendiceal base[1]. Early, the walls of a mucocele may show hypertrophy of the muscle fibers; later, the muscle fibers are absent or thinned, being replaced by connective tissue[4]. Milliken and Poindexter[7] described the formation of intraluminal bodies about the size of small peas. It is believed that these bodies result from inspissated mucus material.

Microscopically, in cases of early mucocele the mucosa may show little change; in older mucoceles, the mucosa cells are flattened or atrophic. The cellular lymphoid components, so abundant normally, are thinned. At first the seromuscular layer may be thickened by inflammation, but later this layer is thinned.

Weaver[2] points out that there is still a question as to whether the cellular response in cases of pseudomyxoma peritonei is neoplastic or inflammatory. Phemister[6] reports a case of ruptured mucocele in which the disseminated intraperitoneal implants disappeared following appendectomy. Apparently removal of the appendix destroyed the source of the implants, and probably the remainder of the implants were absorbed. Experimentally Grodinsky and Rubnitz[4] have shown that in some cases the intraperitoneal transplants disappear. These writers explain this occurrence on the basis of an immunity reaction. However, most authors state that the pseudomucinous lesions eventually become malignant.

Microscopic examination of the implants reveals an outer wall of connective tissue, which may at times be myxomatous. Centrally, there is a necrotic zone containing inflammatory cells, surrounded by a zone of various other cells—eosinophils, giant cells, and large mononuclear cells.

Treatment

The treatment of mucocele is appendectomy. If there has been no leakage, complete recovery is expected; if leakage has occurred and transplants are scattered throughout the peritoneum, it is impossible to remove all the material, and the prognosis is guarded. One has to hope that such a case will be one in which the transplants disappear following removal of the primary focus. Frequently, however, the secondary lesions become malignant. X-ray therapy has been recommended, but its value has not been definitely established.

Report of Cases

Recently we treated a patient with mucocele of the appendix in whom a tumor mass was palpable in the right lower quadrant. A review of the records of Watts Hospital revealed another case in which acute inflammatory changes developed, leading to perforation and peritonitis. A third case was incidentally noted at laparotomy performed for another condition.

Case 1

A 59-year-old white woman was admitted to the hospital September 26, 1946, complaining of a lump in her right side. She had been previously studied and x-rayed in another clinic, where she was told that there was a "mass involving the ascending colon." For the past two years the patient had noted slight pain in the right lower quadrant, which was relieved by lying down. There was no history of nausea, vomiting, or weight loss, and she denied any change in bowel habits. Thirty years previously she had received sanatorium treatment for pulmonary tuberculosis, but follow-up x-rays since that time had been negative. One sister had died with cancer of the breast. The rest of the history was non-contributory.

Physical examination was essentially negative except for a firm, tender, elongated mass in the right lower quadrant, extending from a point midway between the umbilicus and the anterior superior iliac spine upward along the right flank. The mass measured 12½ by 5 cm. It was freely movable.

A complete blood count was within normal limits; urinalysis was negative, except for pyuria. Staphylococcus aureus was cultured from the right kidney. Cystoscopic examination added nothing save for the positive urine culture. The Wassermann and Kahn tests were negative. X-ray examination of the colon with barium revealed "a filling defect in the posterolateral aspect of the cecum." It was smooth in outline, and the appearance suggested an extrinsic tumor mass exerting pressure on the cecum.

The preoperative diagnosis was a cecal tumor or an inflammatory mass resulting from recurrent appendicitis. One observer made a diagnosis of mucocele. After suitable preparation of the patient with

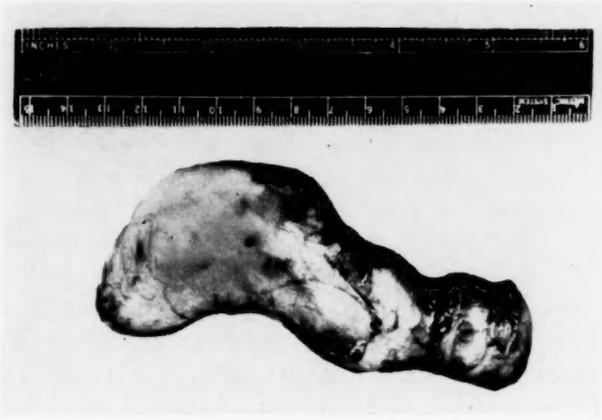


Fig. 1. Mucocele of appendix.

a low-residue diet and Sulfathaladine, the abdomen was opened on October 1, 1946, through a right rectus incision under spinal anesthesia (14 mg. of Pontocaine). A large mucocele of the appendix (fig. 1) was delivered and resected. It had not ruptured. The postoperative course was uneventful, and the wound healed cleanly.

The pathologist's report on the appendix was as follows: "Specimen consists of a "J" shaped appendiceal mass measuring 14 by 3.5 cm. The external surface is smooth, grayish pink, and on palpation, the mass seems to be filled with fluid under pressure. On opening the lumen, thick mucus having the consistency of gelatin is found. The wall is quite thin and fibrous, measuring at one point 3 mm. There is no visible mucosa, the inner surface being smooth. Microscopically, there is complete loss of mucosa. The wall is made up of compressed smooth muscle. Occasional cords of epithelial cells are noted. Diagnosis: mucocele of appendix."

The patient has been followed since her hospital discharge, and she has continued to do well.

Case 2

A 63-year-old white man was admitted to the hospital complaining of generalized abdominal pain of two days' duration. Tenderness and rigidity with rebound tenderness was noted in the right lower quadrant on physical examination. The temperature was 101 F., the pulse 120. The white blood cell count was 15,000, with 86 per cent neutrophils. Urinalysis was essentially negative, and serologic tests for syphilis were also negative.

A diagnosis of acute appendicitis, probably ruptured, was made. The patient was operated on through a McBurney incision, under spinal anesthesia. The appendix had ruptured and was surrounded by a fibrous mass, the size of a small orange. The appendix was removed.

The pathologist reported a "large cyst formation (12 by 7 by 5 cm.) of the end of the appendix. The lumen of the appendix communicated with the cavity of the cyst, and the appendiceal wall was confluent with the sac wall. The microscopic examination revealed acute, diffuse inflammation with suppuration and a purulent exudate. The wall of the sac was composed of inflammatory tissue containing smooth muscle."

Sulfanilamide solution was given subcutaneously, and the patient was discharged fifteen days postoperatively, completely recovered. Recently he was operated on again for another abdominal condition; no trace of any intraperitoneal transplants was found.

Fig. 2. Mucocele of appendix, perforated.

Case 3

A white woman 44 years of age was admitted to the hospital complaining of nervousness and a fullness in her neck, and also of excessive menstrual flow. Physical examination revealed a uterus which was enlarged, nodular, and boggy, and an enlarged, nodular thyroid, the right lobe being chiefly involved. Blood examination showed the hemoglobin to be 58 per cent and the red blood cell count 3,600,000. The urine was negative. Basal metabolism tests were −5 and −11 on two occasions. A chest x-ray and an electrocardiogram were negative. It was decided that a hysterectomy should be done first, because of the menorrhagia and anemia.

The operation was performed under pentothal, gas, oxygen, and ether anesthesia. The uterus was filled with small fibroids, and a mucocele was found in the distal third of the appendix. The pathologic examination revealed leiomyomas of the uterus and a mucocele of the appendix. The microscopic description of the appendix was as follows: "The proximal lumen of the appendix is completely obliterated by scar tissue. In the distal portion the lumen is distended with a mass of mucin. The mucous membrane has been completely destroyed."

Ten days after the abdominal operation subtotal thyroidectomy was done. The postoperative course was uneventful, and the patient was discharged improved on April 24, 1947.

Summary

1. The literature on the subject of appendiceal mucocele is reviewed, and the etiology, pathology, complications, and treatment of this condition are discussed.

2. Three cases of mucocele seen at Watts Hospital are presented.

3. In none of our cases was the major complication of this disease — peritoneal transplants of the pseudomucinous material

—found. From a review of the literature it would seem that some patients with this complication recover completely following appendectomy. However, in the majority of the cases of peritoneal contamination, it is believed that the pseudomucinous lesions eventually become malignant.

Maternal Welfare Section*

REPORT OF PROGRESS OF THE MATERNAL WELFARE COMMITTEE

FRANK R. LOCK, M.D.

WINSTON-SALEM

The Committee on Maternal Welfare was organized in December, 1945. The purpose of the committee is to encourage high standards of obstetric care. In a search for satisfactory methods to reach its objective, the Committee found that an intelligent approach to the problem could not be made until the exact causes of our maternal deaths were determined. A maternal mortality survey was begun on August 1, 1946. This study has been made by requesting, from the physician who signs the death certificate, details of the final illness of any patient whose death is related to pregnancy. Additional information is sought from other physicians who saw the patient during her pregnancy.

A fine spirit of cooperation has been demonstrated by the physicians of North Carolina. Three hundred and ninety-two certificates of death have been sent to the Maternal Welfare Committee by the Division of Vital Statistics of the North Carolina State Board of Health. With two exceptions, the Committee has been given information about the patient's fatal illness by the physician who attended her. In the early months of the study, the barest facts were often sent to the Committee for analysis. Recently, full and detailed records have been submitted in most instances. Minute records of the history, physical findings, and treatment used have allowed an accurate classification and analysis of the deaths. The study of 320 cases has been completed by the Committee, and an analysis of the case record sent to the attending physician.

The studies are made in an effort to give constructive suggestions for the management of similar complications of pregnancy in the future. The Committee fully appreciates the advantage of hindsight, when the full record and result are known to them. They also maintain an idealistic point of view. Many deaths are classified as preventable, even though the members of the Committee fully understand that death was unavoidable under the existing circumstances. Such unavoidable, but theoretically preventable deaths are particularly apt to occur if serious complications develop when a patient is being delivered in a rural home. An increasing tendency to obtain hospital accommodation for patients presenting serious obstetric problems is evident from a review of the Maternal Welfare Committee's records.

The necessity for an educational campaign to reach the poorest and least intelligent people of North Carolina is evident. Forty per cent of the maternal deaths resulted directly from ignorance or neglect on the part of the patient or her family. In these cases, no attempt was made to obtain medical care early enough to save the patient's life, or the patient did not follow the advice when it was given. The Committee is now working toward such an educational program, with the hope of beginning within the year.

Causes of Death (Table 1)

Hemorrhage

Bleeding has now become the leading cause of maternal deaths in North Carolina. Loss of blood was the direct cause of death for 94 patients, and was a major contributing factor in 21 additional cases.

Postpartum hemorrhage was responsible for 55 of these 115 deaths. From the physician's point of view, two grave problems are present: (1) There is a tendency to underestimate or minimize the seriousness of postpartum bleeding. (2) In some cases, a blood loss which seemed moderate resulted in death because of a pre-existing anemia or other causes for lowered resistance. Bleeding often recurs a short time after an initial

Presented to the First General Session, Medical Society of the State of North Carolina, Pinehurst, May 4, 1948.

*Prepared by the Maternal Welfare Committee of the Medical Society of the State of North Carolina:

Frank R. Lock, M.D., Chairman	J. S. Hunt, M.D.
	T. L. Lee, M.D.
J. Street Brewer, M.D.	Ivan Procter, M.D.
G. M. Cooper, M.D.	R. A. Ross, M.D.
E. W. Franklin, M.D.	R. A. White, M.D.

Table 1

MATERNAL MORTALITY SURVEY,
August 1, 1946 - May 1, 1948
(392 maternal deaths reported)

CAUSES OF DEATH IN 320 CASES IN WHICH STUDIES HAVE BEEN COMPLETED*

Hemorrhage — 115 deaths

Hemorrhage was the direct cause of death in 94 cases
Hemorrhage was a contributing cause in 21 cases

Postpartum	55
Rupture of uterus	17
Ectopic pregnancy	14
Abortion	12
Other types	17

78 of these patients received no transfusion

Toxemia — 111 deaths

Convulsive	67
Non-convulsive	44

73 of these patients received poor prenatal care

Infections — 73 deaths

Postpartum	13
Postabortal	17
Intercurrent	21
Tuberculosis	8
Embolism	14

Anesthesia — 15 deaths

Spinal	7
General	8

Heart Disease — 17 deaths

Undetermined and Miscellaneous — 19 deaths

Late Obstetric Complications — 16 deaths

271 were preventable obstetric deaths
13 were non-preventable
36 were non-obstetric deaths

*More than one causative factor was present in several of the cases. These are listed under each of the causes which entered into the fatal outcome.

postpartum hemorrhage is controlled, and extraordinary precautions should always be taken to prevent this occurrence.

Rupture of the uterus with intra-abdominal hemorrhage was responsible for the death of 17 patients. The diagnosis of this condition is difficult, because little or no external bleeding may be present. Palpation and inspection of the lower uterine segment and cervix should be performed in every patient delivered by internal podalic version and extraction. The possibility of rupture of the uterus should be considered in every patient who has had a previous cesarean section or other procedure leading to a scar of the uterine wall.

Facilities to simplify the administration of whole blood by transfusion are urgently needed. A blood bank is an economically sound investment for a small hospital, and numerous such banks must be established to reduce the alarming number of deaths from obstetric hemorrhage. Lack of these facilities accounts for the fact that 78 of the 115 patients who died from hemorrhage were not given whole-blood transfusions. Blood plasma and glucose are temporary measures and will not save the patient's life.

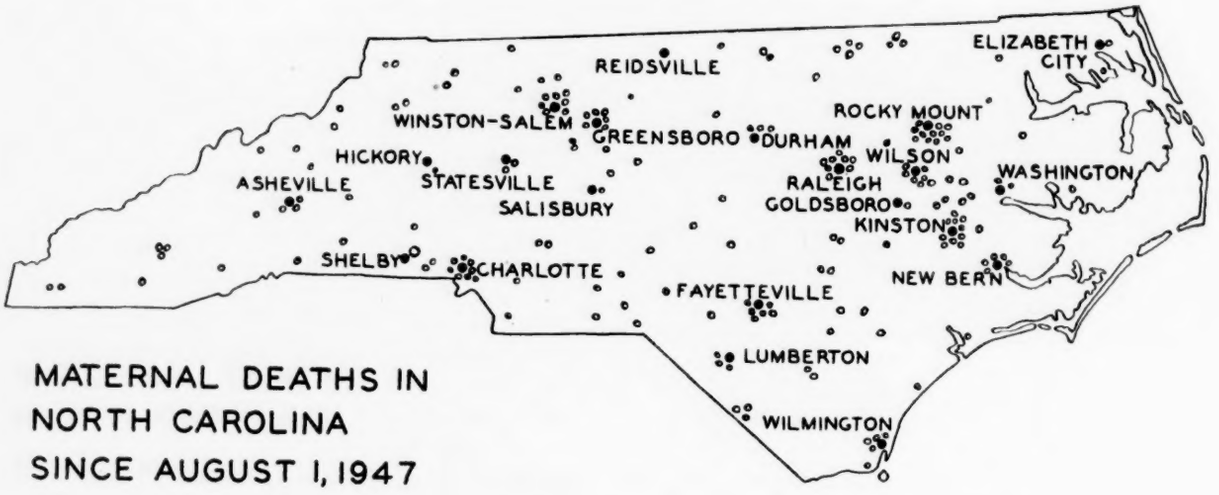
Toxemia

The hypertensive complications of pregnancy continue to be one of the leading causes of maternal deaths in this state. In considering the quality of prenatal care, the Committee noted that many patients failed to return for prenatal examinations according to instructions. In these instances poor prenatal care resulted from indifference upon the part of the patient and her family. Unfortunately, however, there is a tendency upon the part of physicians to consider hypertension during pregnancy in the same category as hypertension in ordinary medical patients. *Blood pressure levels of* 140 *systolic,* 90 *diastolic are an indication of beginning toxemia.* Any slight increase represents a potential hazard to the patient, and hospitalization is indicated for every patient with a blood pressure of 160 systolic, 100 diastolic, or above. Convulsions may occur in patients with blood pressures below this level. Non-convulsive toxemia leading to coma and a progressive downhill course is responsible for nearly as many maternal deaths as is eclampsia.

Infections

The great problem of puerperal infection has almost been conquered by aseptic obstetric technique, and by the use of chemotherapy and antibiotic agents when evidence of infection appears. Postabortal infections are common, however, and treatment may not be requested until the infection is well established. Most cases of embolism are associated with infections of varying severity; and, although an inescapable mortality from embolism is expected, some of the deaths may be avoided by better prophylaxis against infection resulting from abortion and delivery.

The 21 deaths from intercurrent infection have resulted from attempts to treat pneumonia and other serious diseases in the home. Hospitalization is necessary when pneumonia is associated with pregnancy, since its course is more rapid and the mor-



tality higher than in non-gravid patients. Tuberculosis is causing 2½ per cent of our maternal deaths. Only 2 of the 8 fatal cases were diagnosed in the prenatal period, and each of these patients refused to accept satisfactory treatment.

Anesthesia

The fact that 15 deaths resulted directly from anesthetics used for deliveries and operations related to pregnancy is alarming. Spinal anesthesia is particularly hazardous in the presence of toxemia of pregnancy. However, the saddle-block technique, with a hyperbaric solution and a very small dose of the anesthetic agent, was not used for any of the 7 patients who died. Each type of general anesthetic agent has been responsible for one or more deaths. This fact indicates that selection of the anesthetic agent, and skill in its administration are of more importance than the agent *per se*. A careful evaluation of the patient's general condition, transfusions of whole blood to those who are anemic, and selection of special anesthetic agents for patients presenting a complication of pregnancy will materially reduce the number of anesthetic deaths.

Heart disease

Each of the 17 deaths resulting from heart disease was preventable. Early recognition of the presence of a cardiac lesion and careful supervision of the patient are necessary for satisfactory management. Digitalization at the first sign of cardiac failure, and hospitalization with prolonged periods of bed rest may be required. An intercurrent infection in the form of a cold or some other respiratory disease may lead to acute cardiac failure. The closest supervision is indicated under these circumstances.

Undetermined causes of death

In 19 instances, it was impossible to be certain of the cause for the patient's death from the information available. Some of the patients were unattended in the home at the time of death, and the record of a recent medical examination often contained no plausible explanation. In some cases, a definite cause was not ascertained because of an incomplete examination. Too frequently, however, the patient reported to a physician so late in her final illness that there was not time for a complete examination. Under these conditions, autopsies should be performed. Unfortunately, however, postmortem examinations are made infrequently.

Late obstetric deaths

Sixteen patients died some time after their confinement. Seven of the 8 deaths from tuberculosis occurred from six weeks to six months after delivery, bringing the total number of late deaths to 23. In these 7 cases the disease followed a fulminating course after delivery. The remaining 16 deaths resulted from hypertension and its complications, from cardiac disease, and from infections.

Too little attention has been given to this problem of late obstetric deaths. We can hardly consider the survival of a patient at the time of confinement a medical triumph, if she is to expire a few weeks or months later. Furthermore, the progress of a medical condition resulting from pregnancy, such as hypertension due to nephritis, usually shortens the patient's life expectancy. This

fact must be kept in mind in our decisions concerning the management of obstetric cases.

Summary

Three hundred and twenty obstetric deaths have been fully analyzed by the Maternal Welfare Committee. Of this number 271 were preventable if ideal medical care had been available and had been sought by the patient in time.

CHAPTERS IN THE HISTORY OF THORACIC SURGERY

JOSIAH C. TRENT, M.D., F.A.C.S., *Editor*

DURHAM

V

THORACOPLASTY IN PULMONARY TUBERCULOSIS

It is interesting to note that the thorax was the last cavity of the body to be invaded by the surgeon, particularly by the specialized surgeon. Only during the past two decades has the specialty of thoracic surgery arisen—an astounding fact in view of the tremendous strides made in this field during the same period.

Twenty-three years ago Dr. John Alexander[1] analyzed the world results of surgery in the treatment of a disease which had afflicted man for thousands of years. He wrote:

"The surgical management of pulmonary tuberculosis is a relatively new subject . . . Only about 300 cases of pulmonary tuberculosis have been reported as having been operated upon by 17 surgeons in the United States during the past seven years. At the present time there are in this country approximately 30,000 persons with pulmonary tuberculosis who present suitable indications for surgery and who will die of their tuberculosis if they are not operated upon. During the same seven year period only 58 operated cases have been reported from the British Empire, 42 from France, 14 from the Argentine and 2 from Italy. About 1200 cases have been reported by 70 surgeons from Germany, Scandinavia and Switzerland."

The relatively small number of cases reported up to 1925 is rather surprising, since the first operations deliberately undertaken to relax a tuberculous lung by removal of the overlying ribs were performed by de Cerenville[2] of Lausanne in 1885. His article, published in that year, demonstrates that he clearly understood the need for breaking the continuity of the unyielding bony thoracic cage in order to effect collapse of pulmonary cavities. De Cerenville initially resected in 4 cases as much as 3.5 cm. of the second and third ribs anteriorly, or "as many ribs as necessary, according to the extent of the cavity." From this pioneer work our modern thoracoplasty operation has evolved. The results of these first operations by de Cerenville, Quincke, Spengler, and others who followed were unsatisfactory, because the operations were not extensive enough to produce the desired results.

Ludolph Brauer[3] of Hamburg was the first to realize that therapeutic success depended upon obtaining pulmonary collapse comparable to that provided by induced pneumothorax. He proposed, therefore, in place of the limited resections done by de Cerenville, the complete removal of all ribs from the second through the ninth, in order to obtain compression as well as relaxation. Paul Friedrich performed the first operation as suggested by Brauer on December 11, 1917, and the patient survived. The operation was soon modified to include resection of the first and tenth ribs also. It quickly became apparent that this operation was extremely dangerous. Three of the first 7 patients died. Shock, paradoxical respiration, mediastinal flutter, and circulatory embarrassment followed the Brauer-Friedrich operation. Brauer then made several suggestions to overcome the operative dangers, one of which was that the operation be done in stages. The resulting improvement, however, was not sufficient to insure its popularity. Both Brauer and Friedrich made further modifications in the operation later, again without greatly improving the results.

In 1895 Julien Gourdet[4] of Nantes showed experimentally that the size of the thoracic cavity was reduced far more after

1. Alexander, John: Surgery of Pulmonary Tuberculosis, Philadelphia, Lea & Febiger, 1925. This book stimulated the interest of surgeons everywhere in the surgical management of this disease. In 1937 Dr. Alexander wrote his monumental work, "The Collapse Therapy of Pulmonary Tuberculosis" (Springfield, Ill., C. C. Thomas, 1937), which Archibald called the "most comprehensive treatise on the surgical management of tuberculosis in any language."
2. de Cérenville: De l'intervention operatoire dans les maladies du poumon, Rev. méd de la Suisse rom. 5:441, 1885.
3. Brauer, L.: Erfahrungen und Ueberlegungen zur Lungen Kollaps therapie. Die ausgedehnte extrapleurale Thorakoplastik, Beitr. z. Klin. d. Tuberk. 12:49, 1909.
4. Gourdet, J.: Thoracoplastie postérieure. Etude sur l'applatissement comparé du thorax par les différents procédés du resection costale, Paris Institut International de Bibliographie Médicale, 1895.

short paravertebral rib resections than after more extensive lateral, antero-lateral, or anterior rib resections. It fell to Max Wilms[5] of Heidelberg to demonstrate clinically the truth of Gourdet's experimental observations. In 1911 he resected 3 to 4 cm. of the first eight ribs paravertebrally in a tuberculous patient. The patient was markedly improved. This operation, a compromise between the extensive resections of the Brauer-Friedrich operation and the limited resections of de Cerenville, was soon modified so that longer lengths of ribs were removed. After publication of Wilms' work, Ernst Sauerbruch[6] of Berlin advanced his claim to priority for the operation. According to his statement he had performed a paravertebral thoracoplasty in 1909. Both Wilms and Sauerbruch subsequently introduced many modifications of the operation of paravertebral thoracoplasty as originally described by Wilms. Sauerbruch insisted that the lower ribs should be resected first in order to minimize aspiration of infectious secretions by the lower lung during subsequent stages.

The first thoracoplasty for tuberculosis on the American continent was performed in 1911 by Robert G. LeConte at the Pennsylvania Hospital in Philadelphia. In one stage he resected approximately 1½ inches of the paravertebral portions of the first through the eighth rib. The patient was improved. Lambert and Miller[7] in 1924 advocated removing the upper rather than the lower ribs at the first stage of a thoracoplasty, and in 1925 Dr. John Alexander and others began to remove increasingly great lengths of the uppermost ribs in multiple stages. Ochsner[8] and Hedblom[9] first emphasized the greater safety afforded by multiple stages rather than a one- or two-stage procedure. Thus our modern thoracoplasty has become a multiple-stage operation in which great lengths of the ribs, as well as the transverse processes, are removed over the diseased portion of the lung from above downwards.

5. Wilms, M.: Eine neue Methode zur Verengerung des Thorax bei Lungen tuberkulose, München. med. Wchnschr. 58:777, 1911.
6. Sauerbruch, E., quoted by Alexander, J., (1), p. 34.
7. Lambert, A. V., and Miller, J. A.: The Surgical Treatment of Pulmonary Tuberculosis, Tr. Assoc. Am. Phys. 39:112-119, 1924.
8. Ochsner, A. J.: Discussion of Archibald, E.: Pulmonary Tuberculosis; Its Surgical Treatment, J.A.M.A. 85:663-668 (Aug. 29) 1925.
9. Hedblom, C. A.: Extrapleural Thoracoplasty in Pulmonary Tuberculosis, J.A.M.A. 86:374 (Jan. 30) 1926.

In 1925 Dr. Alexander "received a letter from a distinguished physician whose hospital connections included professorships in two University hospitals in one of the largest cities in the United States. He wrote as follows: 'My surgical and medical friends here both agree that they have never seen the slightest benefit from the operation of paravertebral thoracoplasty and they do not recommend it in any case.' "[10] In the short span of twenty-three years this sage opinion has become invalid. Today thoracoplasty is a safe, effective procedure for the control of pulmonary tuberculosis and is performed daily all over the world, restoring to normal, active life many patients otherwise hopelessly ill.

J. C. T.

10. Alexander, John(1), p. 19.

"Detail Man"*

Personally, we like most "detail men." In case you haven't had contact with one recently, may we refresh your mind on the subject. Were we Linnaeus, we might describe him thus: Genus: Homo Sapiens; Habitat: Distribution almost universal, but becoming scarcer in Middle West due to the draft and lack of enforcement of game laws. (Most Dox think there is a perpetual open-season on these chaps.) Description: A hardy perennial. (Webster's definition of perennial: "continuing or enduring through the year or many years.") And, Boy, does he continue to endure a lot!

Further description: This sub-order of Homo Sapiens not infrequently is married and sires one or more little detailettes who depend upon the parent shrub for food and raiment. He has the customary complement of manual and pedal appendages; also, two ears, two eyes, two lungs and—believe it or not—a heart.

Usually he is a gentleman, which in itself is saying a lot. Obviously this rare specimen has an inexhaustible fund of patience, otherwise he would not be willing to cool his heels in your reception room for long periods of time, awaiting your willingness and readiness to see him for five minutes. He knows, of course, that in order to impress him with your importance, he will have to sit on his quadriceps in the outer sanctum until you get darn good and ready to admit him to your august presence . . .

So if you see one of these roving, self-abnegative, hard-working, patient and pleasant fellows beginning to take root in your reception room, for Heaven's sake have the girl bring him in before he becomes a permanent potted plant before your very eyes. Because all of you know how much easier it is to dispose of cut flowers, than a pardiniere full of flowering hydrangeas.

But seriously, Fellows, let's give these boys a break. We are busy, of course, but not too busy to spare a few minutes of our time when it easily might be of mutual benefit. Ever hear of the Golden Rule? Think it over sometime; it will do you good.

—J. Phil Edmundson, M.D.

* The Jackson County Medical Society Weekly Bulletin, March 4, 1944; reprinted in J. Arkansas M. Soc. 44:244 (April) 1948.

North Carolina Medical Journal

Owned and published by
The Medical Society of the State of North Carolina,
under the direction of its Editorial Board.

EDITORIAL BOARD

Wingate M. Johnson, M.D., Winston-Salem
Editor

Miss Catherine Johnson, Winston-Salem
Assistant Editor

Roscoe D. McMillan, M.D., Red Springs
Business Manager

Paul H. Ringer, M.D., Asheville, Chairman
W. Reece Berryhill, M.D., Chapel Hill
Coy C. Carpenter, M.D., Winston-Salem
Ernest W. Furgurson, M.D., Plymouth
Hubert A. Royster, M.D., Raleigh
Josiah C. Trent, M.D., Durham

Address manuscripts and communications regarding editorial matter to the
NORTH CAROLINA MEDICAL JOURNAL
300 South Hawthorne Road, Winston-Salem 7, N. C.

Questions relating to subscription rates, advertising, etc., should be addressed to the Business Manager, Red Springs, N. C.

All advertisements are accepted subject to the approval of the Council on Pharmacy and Chemistry of the American Medical Association.

Annual subscription $3.00 Single copies, 30¢

Publication office: Carmichael Printing Co., 118 West Third Street, Winston-Salem 1, N. C.

MAY, 1948

THE NINETY-FOURTH ANNUAL SESSION

For its ninety-fourth annual session the Medical Society of the State of North Carolina came back home to Pinehurst. Although Virginia fully lived up to her reputation for hospitality last year, Virginia Beach was a long way from home for most of our members. Last year's attendance was the smallest—except for the Bermuda cruise meeting—since 1924.

This year's registration broke all previous records, with a final score of 920 doctors registered—31 more than attended the 1946 meeting. The exhibits, both technical and scientific, were housed in a tent, connected by a covered walk with the Carolina Hotel. The exhibitors, who were not at all happy over the situation at first, were more than satisfied before the meeting was over. They had much more room in the tent than they had had in the hotel; their wares could be displayed to better advantage; and the doctors kept the tent comfortably filled most of the time, and overflowing often. The twelve prizes offered—headed by a General Electric refrigerator, which was won by Dr. George Harrell of Winston-Salem—no doubt helped stimulate the attendance on the exhibits. Mr. J. T. Barnes, our new executive secretary, deserves much of the credit for the successful handling of the exhibits.

The House of Delegates, as usual, had a full program; but there was a surprising lack of controversy in its meetings. The absence of Dr. Frank Sharpe was felt throughout the session, but Dr. J. F. Robertson made an excellent presiding officer. His address to the House, although brief, was full of meat. The most important of his recommendations—to raise the annual dues from $10 to $25—was passed with very little opposition. The majority of the members realized that the Society is undertaking a program of expansion that will require a greatly increased budget.

Another recommendation which met with general approval was that the by-laws be changed so that the first vice president, rather than the president-elect, would fill the unexpired term of a president dying in office. This custom is followed by other organizations, and is the logical thing to do. One year is long enough to ask any doctor to serve as president of the society; furthermore, it is a somewhat awkward situation for the president-elect to succeed himself as president, rather than to be installed by the retiring president.

The scientific program was good, and well attended. One of the guest speakers who appeared before the general sessions—Dr. J. deJ. Pemberton of the Mayo Clinic—is a native of Fayetteville. He gave an excellent address on thyroid surgery. Dr. Russell Haden, of Cleveland, spoke before the second general session on the anemias, especially the treatment of pernicious anemia. Many afterward said that the classification of the anemias and the treatment of pernicious anemia could not possibly have been made plainer or more practical. Dr. William Friedenwald, of the Department of Bacteriology at Emory, spoke on poliomyelitis. His talk, too, was interesting and informative.

Something of an innovation this year was the panel discussions held by various sections. Even the Officers' Breakfast on Tuesday morning was devoted to a panel discus-

sion of medical organization problems. The sections on Practice of Medicine, Neurology and Psychiatry, General Practice, and Radiology all used the panel system.

The President's Night banquet was featured by the paper of Mr. James H. Clark on the Medical Care Program and a humorous address by Mr. James E. Gheen, of New York. Dr. Oren Moore as toastmaster, with Secretary McMillan as master of ceremonies, ensured the success of the occasion.

The report of the Nominating Committee apparently met with the approval of the great majority of the society. Dr. Westbrook Murphy of Asheville, the president-elect, has earned his recognition by the consistently high quality of the service he has rendered the society and organized medicine. The other officers are as follows: Dr. J. J. Combs, Raleigh, first vice president; Dr. J. A. Elliott, Charlotte, second vice president; Dr. Roscoe D. McMillan, Red Springs, secretary-treasurer.

* * * *

THE AMERICAN ACADEMY OF GENERAL PRACTICE

Faithful readers of this journal may recall that last July most of an editorial on the general practitioner[1] was devoted to a discussion of the American Academy of General Practice, which was organized at the centennial meeting of the American Medical Association. Since then this organization has grown steadily. Some concern was felt at first lest it should become a secession from the A.M.A., or should degenerate into a political pressure group. Representatives from the Academy, however, had a most amicable meeting with the Board of Trustees of the A.M.A. last February. They assured the Board that leaders of the Academy were anxious to cooperate with the parent organization, and asked that representatives from the Board, from the Council on Medical Education and Hospitals, and from the Council on Medical Service be appointed as a liaison committee to the Academy. This request was granted, and there is no reason to doubt that the Academy can do much to carry out its avowed purpose "to promote and maintain high standards of the general practice of Medicine and Surgery."

1. The General Practitioner Has His Innings, Editorial, North Carolina M. J. 8:420 (July) 1947.

A chapter of the American Academy of General Practice has been organized in North Carolina, with Dr. John R. Bender of Winston-Salem as president, Dr. W. A. Sams of Marshall as vice president, and Dr. Roscoe D. McMillan of Red Springs as secretary and treasurer. District councilors are: Dr. John A. Payne, Sunbury, First District; Dr. G. Grady Dixon, Ayden, Second District; Dr. Amos N. Johnson, Garland, Third District; Dr. Henderson Irwin, Eureka, Fourth District; Dr. L. R. Doffermyre, Dunn, Fifth District; Dr. Charles A. Bland, Louisburg, Sixth District: Dr. W. E. Selby, Charlotte, Seventh District; Dr. A. D. Ownbey, Greensboro, Eighth District; Dr. I. E. Shafer, Salisbury, Ninth District; Dr. V. H. Duckett, Canton, Tenth District.

An application blank is to be found following page 280, for the convenience of those who wish to join this organization.

* * * *

EDITORIAL NOTES—THE NINETY-FOURTH ANNUAL SESSION

The panel discussions used by a number of the sections were interesting, and apparently were liked by most of those in attendance. It remains to be seen what their effect upon the NORTH CAROLINA MEDICAL JOURNAL will be. If all those who participated in the panels wrote out their discussions and turned them in to the secretaries of the sections, they should make interesting reading during the year; but if the participants spoke from notes not prepared for publication, this journal may have to stretch its material to make it last through the year.

* * *

Last year, for the first time, the president's address was the feature of the President's Night banquet. This year the precedent was not followed. Since President James Robertson succeeded himself as president, he decided to reserve his speech until next year. Since he will have had eighteen months to prepare his address for the 1949 meeting, the members will expect a real masterpiece.

* * *

The large number who attended the Conjoint Session of the Society and the State Board of Health to hear the final report of Dr. Carl Reynolds as State Health Officer was a tribute to Dr. Reynolds. It is not surprising that Dr. Reynolds was too overcome

with emotion to finish his report, for many of his audience were close to the same point. Under his leadership the State Board of Health has made remarkable progress, as was shown by his report. The best wishes of the doctors of North Carolina go with him as he prepares to show Californians how a model grandfather should conduct himself.

The brief inaugural address of Dr. Roy Norton, who is to succeed Dr. Reynolds as State Health Officer, was a masterpiece, and made a profound impression on his audience. It is the general impression that his selection was a wise choice.

* * *

There was an altogether different atmosphere in the Carolina Hotel this year, as compared with 1946. Even though the registration was larger, the management of the hotel did not seem so hurried and harried; the hotel employees seemed friendlier and more courteous; and the food was better. It must be admitted, also, that the conduct of the members left much less room for criticism. Indeed, the head waiter told Dr. McMillan at lunch Wednesday, in the presence of a reliable witness, that he had never handled a better behaved convention. He did not qualify the statement by saying "medical convention," but "convention"—period!

* * * *

"LET NOT THY LEFT HAND KNOW . . ."*

The United States Public Health Service certainly follows the old Biblical injunction. As followers of these columns know from recent issues, the distinctly "left" hand of the U.S.P.H.S. and other federal bureaus are marching up and down the land—and even visiting foreign lands—stumping for compulsory federal sickness insurance and government control of medicine, using arguments based upon the supposedly deplorable state of the nation's health. My, Oh My, how sick the nation is, to hear the left hand speak. Goodness me, how the medical profession has failed. It's terrible, how a third of our population can't find a doctor when one is wanted—and how they can't pay him if they do find him.

Now comes the right hand—of the self-same U.S.P.H.S.—in a statement to the press that "the general health of the people of the United States, maintained during the war years at a *higher than prewar level*, continued favorable during the first six months of 1947" quoting Federal Security Administrator Oscar R. Ewing. (Italics ours.) It goes on: "The reported incidence of most of the important communicable diseases, the crude death rate, the maternal and infant mortality, and the specific death rates for certain diseases indicate as good health conditions in the United States during the first half of 1947 as in 1946, if not slightly better." Yes, this is quoted directly from the U.S.P.H.S.

The statement then goes into considerable statistical detail. Since the left hand has screamed so loudly about infant and maternal mortality from the Children's Bureau whose appropriations must go up and up every year if such bureaus are to be supported in the style to which they would like to become accustomed, let us see what the right hand says about the cold facts for 1947. Again, direct quotes from the U.S.P.H.S.: "The estimated maternal mortality rate for deaths from puerperal causes was 1.5 per 1,000 live births for the first six months of 1947, as compared with 1.7 for the corresponding period in 1946; and the adjusted infant mortality rate was estimated to be 34.6 per 1,000 live births for the first six months of 1947, as compared with 38.4 in 1946."

Of course there is plenty of room for improvement; no scientific man would deny it. The medical profession will lower future mortality rates, is constantly doing so, and neither the medical profession nor the great American public needs to be socialized or communized or nazified to do it. Sometimes we wonder how men with the scientific training that Parran, Mountain, Hilleboe, et al, must have had can help but choke on their words when on the one hand they reveal the facts of our constantly improving health standards and on the other they propagandize the country for a socialistic scheme to "improve" health that has wrecked the health of every nation that has let the bureaucrats take over. Perhaps they forget that even the Bible, quoted out of context, can lead to gross misinterpretation.

*Reprinted from the Rocky Mountain Medical Journal 45: 19-20 (Jan.) 1948.

Clinicopathologic Conference

BOWMAN GRAY SCHOOL OF MEDICINE
OF WAKE FOREST COLLEGE

A 7-month-old white female infant was admitted to the North Carolina Baptist Hospital on November 29, 1947, with chief complaints of pneumonia, fever, cough, cold, and poor appetite. Two months before admission the patient began to have a runny nose, with sneezing and cough. Her appetite at this time was good. Six weeks before admission the local physician was called and made a diagnosis of pneumonia. The patient was given penicillin orally, one half tablet every three hours, and seemed to improve for a while. When the penicillin was discontinued two days before admission, her condition grew worse. She seemed to be weak and was more irritable. Her appetite became much poorer, but she continued to drink some milk. The cough improved in spite of the patient's weakness. On the day of admission she became worse, and about six hours before admission she began to breathe with difficulty.

Physical examination showed the temperature to be 103 F., the pulse 160, respirations 80. The baby was thin and undernourished. Her skin was dry and pale, and breathing was rapid and shallow. The patient was listless and only slightly responsive. The anterior fontanelle was patent, but there was no craniotabes. The eyes were sunken. Both ear drums were reddened and injected, especially the right; there were no distortions of landmarks, however. The lips and mucous membranes were dry. The tongue was slightly coated and the pharynx was moderately injected. Retraction of the interspaces was noted on inspiration. Moist, sticky rales were heard over the lung fields posteriorly, especially on the right; rales were also heard over the anterior portion of the chest. There were no evidences of consolidation, and no impairment of breath sounds or resonance. The heart was rapid and the pulse difficult to count, but there were no detectable arrhythmias or murmurs. The liver was felt one finger's breadth below the costal margin in the midclavicular line. The spleen was not palpable. No masses were felt in the abdomen. The genitalia were normal, and rectal examination was not remarkable. There was slight cyanosis of the nails; otherwise the extremities were well developed without deformities. Reflexes were physiological.

A blood count showed 4,890,000 red blood cells, with 14 Gm. of hemoglobin, and 52,750 white blood cells with 24 per cent segmented polymorphonuclears, 54 per cent non-segmented, and 22 per cent lymphocytes. The urinalysis showed a specific gravity of 1.009 and a negative reaction for albumin and sugar; microscopic examination showed an occasional white cell and epithelial cell in the sediment. The stools were yellow, formed, and had a foul odor. Numerous fat globules were seen in the stool, but no starch or occult blood was present. The blood culture was sterile. A throat culture revealed beta hemolytic streptococci, non-hemolytic staphylococci, and coliform organisms. Trypsin digestion tests on duodenal fluid showed no liquefaction of the gelatin at the end of twenty-four hours. The Schick and tuberculin tests were negative.

An x-ray of the chest revealed a marked increase in bronchovascular markings in the hilar region, extending out into the periphery of the lung field. The extreme periphery of the lung fields was clear. The heart showed nothing remarkable.

During her hospital stay of fifteen days, the patient was treated with penicillin intramuscularly. On the fifth hospital day, streptomycin was begun and was continued through the eleventh hospital day. Penicillin aerosol was begun on the fourth hospital day and continued through the tenth hospital day. She was also given frequent intravenous injections of fluids, including plasma and whole blood. In spite of this therapy her condition remained critical. At no time did she show any evidence of diarrhea or disturbance in bowel function. During the first week the temperature ranged between 98 and 101 F., and during the second week it ranged between 99 and 102 F. She expired quietly on the fifteenth hospital day.

Discussion

DR. WESTON M. KELSEY: This protocol can be summarized rapidly. The infant was apparently well until the age of 5 months, when she developed bronchopneumonia which persisted for two months in spite of

treatment with antibiotic and chemotherapeutic agents. The important accessory clinical findings were the presence of microscopic neutral fat in the stools, and the absence of trypsin in the duodenal juice. The patient went down hill rapidly and died. Since these findings make the diagnosis of "cystic disease of the pancreas" obligatory, this case will be used to discuss the diagnostic problems in this disease.

In general, the history obtained at the time of admission depends on the age of the patient. Andersen[1] has pointed out that these cases may be divided into three categories: (1) those which begin at birth; (2) those which begin before the age of 6 months; and (3) those in which the symptoms develop at a later age. The first group are nearly always characterized by intestinal obstruction with meconium ileus. In approximately one third of Andersen's cases which fell in the second group, the first sign of trouble was cough; in the remaining two thirds there were gastrointestinal symptoms, which often were minimal and were described only after specific questioning. In the last group the majority of patients presented the picture of "celiac disease," with the typical large, foamy, foul stools.

Blackfan and May[2] state that the early symptoms of cystic fibrosis can mimic almost any disease seen in the second age group. Vomiting, feeding problems, and failure to gain are almost always noted before there is either diarrhea or cough. I am almost certain that a review of this case will reveal that feeding problems were present before the onset of pneumonia.

DR. ADERHOLDT: This patient was difficult to feed and had failed to gain at a normal rate.

DR. KELSEY: The usual findings on physical examination are those noted in this patient. The infant is malnourished. The respiratory symptoms usually predominate, and are characterized by a hard cough which is similar to that seen in whooping cough. Frequently there is no evidence of consolidation. The rales are coarse and are heard over the entire chest. Cyanosis is common in the more severe cases.

Laboratory studies are necessary to establish the diagnosis. Of these the most important are examination of the stool and analyses of the duodenal enzymes. The stools may be small or large, loose or constipated, or normal in gross appearance. Regardless of the appearance, they usually have a peculiar, foul odor. The presence of fat in the stool may be determined by either chemical or microscopic study. The former method is not practical, while the latter is simple. Andersen[3] has described the criteria for determining microscopically the presence of excessive fat in the stools. The results of her determinations correlated closely both with chemical studies, and with postmortem and clinical observations.

There are two relatively common conditions which cause an increase in neutral stool fat: celiac disease and obstructions of the common bile duct. The rare causes of this condition are intestinal allergy, giardiasis, tuberculous mesenteric lymphadenitis, and intestinal lipodystrophy. False positive tests are seen in patients who have received mineral oil. False negative tests occur if the patient is on a fat-free diet. Andersen makes the statement that "absence of fat on a normal diet excludes fibrocystic disease." In all but one of the 15 patients with fibrocystic disease whom we have seen, excessive fat in the stools was demonstrated by this method.

Our experience would indicate that the presence of twenty or more globules of fat per low-power field on repeated examinations certainly warrants duodenal drainage. Ordinarily the tube enters the duodenum easily, but at times it must be manipulated under the fluoroscope. If the patient has fibrocystic disease, the juice is usually scant and viscid. It should be bile-stained and have a pH above 6.5. Analysis for trypsin is done according to the technique of Andersen[4]. It is possible for an error to result from failure to have the tube in the duodenum. Tests which fail to demonstrate trypsin should be repeated. Theoretically, tests which are positive for trypsin cannot be in error, and they need not be repeated. The presence of trypsin rules out the possibility of fibro-

1. Andersen, D. H.: Cystic Fibrosis of the Pancreas and Its Relation to Celiac Disease: A Clinical and Pathologic Study, Am. J. Dis. Child. 56:344-399 (Aug.) 1938.
2. Blackfan, K. D., and May, C. D.: Inspissation of Secretion, Dilatation of the Ducts and Acini, Atrophy and Fibrosis of the Pancreas in Infants: Clinical Note, J. Pediat. 13:627-634 (Nov.) 1938.
3. Andersen, D. H.: Celiac Syndrome: Determination of Fat in Feces, Am. J. Dis. Child. 69:141-151 (March) 1945.
4. Andersen, D. H.: Pancreatic Enzymes in the Duodenal Juice in the Celiac Syndrome, Am. J. Dis. Child. 63:643-658 (April) 1942.

cystic disease. Absence of trypsin on repeated drainages has been described in only one patient who did not have the disease[5], and makes the diagnosis almost certain.

The diet and treatment of the pulmonary infection are the two important aspects of therapy. The diet should be high in protein and carbohydrate, and low in fat and starch. Pancreatin occasionally will help. The intake of vitamins A and D should be considerably increased[6]. The bronchial infection is usually treated most satisfactorily with penicillin aerosol[7]. These patients are surviving for much longer periods than before the above mentioned therapeutic measures were used, and it is possible that the prognosis might be good if the diagnosis were made before the onset of respiratory symptoms. The prognosis is still poor in the infant with an early onset of pulmonary disease.

The etiology of fibrocystic disease is not known. It is a congenital and familial disease. Six siblings of the fifteen patients we have had with this disease have died with symptoms compatible with the diagnosis of fibrocystic disease. I have asked Dr. Herndon to discuss the hereditary aspect.

DR. NASH HERNDON: Although several authors[8] have reported multiple cases of cystic fibrosis of the pancreas within a sibship, the most adequately described series of cases collected in a manner suitable for genetic analysis is that of Andersen and Hodges[9]. They reported all cases diagnosed at the Babies Hospital in New York from 1938 to 1945, including data on 60 patients. An analysis of the familial incidence in this group gives evidence that the disease may be due to the action of a simple recessive gene, and that the condition develops in individuals who are homozygous for this gene. Data on 55 other sibships collected from the literature are also compatible with this conclusion.

The question of the incidence of this disease in the general population is of considerable interest. The incidence reported in autopsy material varies from 1.3 per cent to 4.8 per cent, about 3 per cent being the usual figure given[10]. Andersen has estimated the incidence of the disease to be around 1.7 to 1.8 per thousand live births in New York state. There are obvious difficulties in estimating the frequency of a disease from a series of hospital admissions or an autopsy series. However, a very rough estimate of the number of persons who are heterozygous for the recessive gene—"carriers" of the disease—can be made from the above figures, and is found to be around 8 per cent of the population. One would then expect that about six marriages per thousand would represent the mating of two heterozygotes, and might produce children with the disease. Because of the small size of human families and the fact that only one fourth of the children of such marriages would be affected, any clinical survey would make it appear that the proportion of families affected is considerably smaller. This estimate of a rather high incidence of the disease in the population is also supported by the absence of cousin marriages among the parents of patients in Andersen's series, and the fact that only one case report in the literature reports kinship of the parents[11]. In diseases due to recessive genes, the frequency of consanguineous matings among the parents of affected individuals is inversely related to the frequency of the disease—or, more specifically, to the frequency of the gene; in extremely rare diseases the rate of cousin marriages among the parents of affected individuals may be as high as 50 per cent. The lack of cousin marriages in the reported cases confirms the impression that the disease is much more common than one would gather from the number of diagnoses made. As physicians become more familiar with the syndrome, this disease will probably be diagnosed during life much more frequently.

DR. KELSEY: The diagnosis is not quite so easy as has been implied. Three of our cases illustrate some of the problems. Baby H. was admitted at 3 months of age because of failure to gain. He had had one fourteen-day

5. Maddock, C. L., Farber, S., and Shwachman, H.: Pancreatic Function and Disease in Early Life, Am. J. Dis. Child. 66:370-375 (Oct.) 1943.
6. Andersen, D. H.: Celiac Syndrome; Dietary Therapy for Congenital Pancreatic Deficiency, Am. J. Dis. Child. 70: 100-113 (Aug.) 1945.
7. di Sant'agnese, P. E. A., and Andersen, D. H.: Celiac Syndrome; Chemotherapy in Infections of the Respiratory Tract Associated with Cystic Fibrosis of the Pancreas: Observations with Penicillin and Drugs of the Sulfonamide Group, with Special Reference to Penicillin Aerosol, Am. J. Dis. Child. 72:17-61 (July) 1946.
8. Howard, P. J.: Familial Character of Fibrocystic Disease of the Pancreas, Am. J. Dis. Child. 68:330-332 (Nov.) 1944.
9. Andersen, D. H. and Hodges, R. G.: Celiac Syndrome; Genetics of Cystic Fibrosis of the Pancreas with a Consideration of Etiology, Am. J. Dis. Child. 72:62-80 (July) 1946.
10. Wiglesworth, F. W.: Fibrocystic Disease of the Pancreas, Am. J. M. Sc. 212:351-365 (Sept.) 1946.
11. Garrod, A. E., and Hurtley, W. H.: Congenital Family Steatorrhea, Quart. J. Med. 6:242-258 (Jan.) 1913.

episode of watery diarrhea when 6 weeks old, and the stools had always been foul. The stools did not have excessive fat, but two duodenal drainages failed to reveal any trypsin. He could be one of the rare patients without cystic fibrosis who does not have demonstrable trypsin. Baby A. was admitted because of convulsions, and was noted to have foul stools. There was excessive fat, and no trypsin was found. She has done well over a twenty-month period. It has been found that the only dietary control necessary for adequate growth is pancreatin. Without pancreatin, the stools become characteristic of celiac disease. She has had no pulmonary complications. Because of the doubt as to the diagnosis, she has had five drainages, all negative for trypsin. Baby B. was admitted at 10 months of age with a history typical of the disease. Excessive fat was demonstrated in the stools, but, to our surprise, trypsin was present in moderate amounts. She went down hill rapidly, and died from advanced bronchiectasis. Autopsy was not obtained.

We feel that either repeated pulmonary infections or chronic gastrointestinal disturbances in patients under a year of age warrant examination of the stools for fat. If this is found, we feel that duodenal drainage should be performed. If excessive fat is present in the stools and trypsin is absent in the duodenal juices, the diagnosis of cystic fibrosis of the pancreas must be made.

Anatomic Discussion

DR. LADD W. HAMRICK, JR.: The significant anatomical findings were in the pancreas, lungs, and liver. The pancreas weighed 15 Gm. and presented no gross abnormalities, except for slightly increased resistance on sectioning. Microscopic examination showed dilatation of the majority of the acini and ducts, with atrophy of the lining cells; the lumina of these structures contained pink-staining laminated concretions. The interstitial connective tissue was increased, and a moderate degree of interstitial lymphocytic infiltration was present.

The lungs presented fibrous pleural adhesions on their posterior surfaces, and many small, closely adjacent areas of consolidation on cut section. The bronchi contained large amounts of yellow, purulent material. Microscopic examination showed both acute and chronic bronchopneumonia, marked interstitial fibrosis, and chronic bronchitis with destruction of many of the bronchial walls. Culture of the lungs revealed a pure growth of Staphylococcus aureus.

The liver showed fatty metamorphosis.

Anatomic Diagnoses

1. *Cystic fibrosis of the pancreas*
2. Bronchopneumonia, acute and chronic, bilateral (Staphylococcus aureus)
3. Chronic bronchitis with bronchial destruction, bilateral
4. Chronic interstitial pulmonary fibrosis, bilateral
5. Chronic fibrous pleuritis, bilateral
6. Fatty metamorphosis of the liver

MEDICOLEGAL ABSTRACT

J. F. OWEN, M.D., LL.B.
RALEIGH

MASTER AND SERVANT: *Neurosis which results from an injury arising out of and in the course of employment, and produces disability, is compensable, whether of functional or of organic origin.*

The records show that the plaintiff in this case suffered personal injuries from an accident which arose out of and in the course of her employment. While she was sharpening razor blades, pieces of a broken blade, propelled from a machine employed in the sharpening process, struck her in the right eye, on the nose, about the right arm, and on the little finger of the right hand. The employee suffered an extensive horizontal laceration of the cornea, and, despite immediate surgical intervention, she lost the vision in the injured eye. A medical examination disclosed some tremor, hyperactive deep reflexes, and unsteadiness of gait. She lost weight and suffered from fainting spells. The subjective symptoms were pain in the arms and right hand, weakness of the right hand, headaches, vertigo, insomnia, and somnambulism. The employee was awarded total compensation for the loss of the eye, and 10 per cent of total permanent disability for impairment due to a neurosis.

The commissioner hearing the case was of the opinion that there was substantial basis for a finding that the physical injuries,

other than the laceration of the eye, contributed to the plaintiff's neurotic condition. Further, it was brought out that she had had marital difficulties, and her physician testified that as a result she was fertile ground for the development of a psychoneurosis. The physician, an expert neurologist, testified, however, that "trauma was unquestionably the exciting and precipitating cause of the neurosis," and that "superimposed psychoneurosis certainly adds to the disability (that resulting from the loss of vision)."

From the findings of the Workman's Compensation Commission the defendant appealed to the appellate court. In doing so he conceded that there was evidence to sustain the finding that the neurosis was a result of the accident, but insisted that it was a disability incident to the loss of the eye, and that compensation therefor was included in that prescribed for the loss of vision in the injured eye. The appellate court did not hold with the appellant that the injury to the eye was the sole cause for the development of the neurosis. The court indicated that in its opinion the evidence was sufficient to show that other physical injuries contributed to the production of the emotional disorder, and that the marital difficulties noted above must be considered predisposing factors. In explanation the justice writing the opinion said, "the construction contended for by the appellant, if carried to its logical conclusion, would deny compensation for both eyes if the sight of the left eye were destroyed as a result of a sympathetic ophthalmia, consequent upon an injury to the right eye. The loss of the left eye from sympathetic affection, if that occurred, would be a mere incident of the injury to the right eye. And there could be no recovery for the disability resulting from the consequent neurosis, even though, in its paralysis of bodily function, it was a disability that far exceeded the loss of an eye in its disastrous consequences to the victim."

From the above case we may assume that the law recognizes emotional disorders, such as the neuroses, as being disease entities and compensable to the same extent as similar disabilities occurring in organic diseases. Other courts are in agreement. Because of the prevailing opinion of many laymen that emotional disorders are purely imaginary it has, in the past, been very difficult for those suffering from this type of illness to secure the rights to which they are legally entitled. Difficulties in this respect occur not only in matters actually before the courts but in informal negotiations with insurance companies, employers, and others. It seems, therefore, that when a patient has a meritorious cause of action based upon a disability arising from an emotional disorder, it is the duty of his physician to establish or to help establish the facts in such a manner as to protect the patient. In such cases the conscientious efforts of the trained physician will do more than anything else to ensure justice to all concerned.

(Court of Errors and Appeals of New Jersey, May, 1933. V. 166, Atlantic Reporter, p. 518.)

TUBERCULOSIS ABSTRACTS

A Review for Physicians

Issued Monthly by the National Tuberculosis Association

Vol. XXI May, 1948 No. 5

THE economic importance of controlling tuberculosis is often overlooked because the humanitarian aspect is so compelling. Therefore, it is good to be reminded that it is business organizations, established for profit, which have furnished much of the clear-cut unmistakable evidence that the control of tuberculosis is both possible and practical with the means now at our disposal.

TUBERCULOSIS CONTROL IN INDUSTRY

Tuberculosis control among employees of The Eastman Kodak Company was begun in 1921 with a roentgenographic survey of the chests. In 1923 it was enlarged to include periodic roentgenograms of the chests of all employees as well as the examination of applicants.

In a report made by Dr. William A. Sawyer in 1939, this work was reviewed for the years 1923 to 1937. At that time the incidence of active disease per thousand employees in three-year periods was shown to have dropped from 2.08 in the period 1923 to 1925 to 0.47 in the period 1935 to 1937. During the war years the labor turnover was unusually high, and therefore the incidence cannot accurately be determined. In only 18 persons did active pulmonary tuberculosis develop over a period of five years, among employees increasing in number from 6,000 in 1941 to 10,000 in 1945. Though the increment was only 4,000, the number of applicants employed for varying periods of time during the period 1941 to 1945 totaled 20,500. Active pulmonary tuberculosis appeared at all age levels. The ratio of the number of cases in each decade to the percentage in each ten-year group employed is nearly uniform throughout.

Those in the higher age groups might even be regarded as more vulnerable to tuberculosis, since

they have been subject to a "weeding out" process over the years. Those who survive have the same attack rate as those aged 20 to 40 years, usually considered to be more vulnerable.

At the present time two per cent of all employees have roentgenograms of the chest classified as indicating pulmonary tuberculosis, minimal inactive.

Following the preemployment roentgenogram, routine 14 by 17 inch roentgenograms are retaken as follows:

Age When Employed	*Years Between Routine Roentgenograms*
To 25 years	1, 3 and 5
25-34 years	2, 3 and 5
35 to retirement	3 and 5 and every 5 years thereafter

In addition roentgenograms are taken after prolonged absence for any reason, and in the presence of suggestive symptoms.

The majority of the group (11 cases) in which active pulmonary tuberculosis developed later had roentgenograms of the chest indicating abnormalities at the time of employment. In three cases this was an "apical cap," a term used to describe a crescentic, homogenous soft tissue density, over the dome of the apex of the lung.

Stage of Disease When Discovered to be Active

The one case of far advanced disease discovered contradicts an oft repeated rule, namely, that a person with a normal roentgenogram of the chest at age 40 will never have active pulmonary tuberculosis. This woman, aged 56 at the time of employment, had what was considered to be an inactive infraclavicular lesion. One year after employment she was taken acutely ill with what appeared to be virus pneumonia. Three weeks after onset of the disease her sputum contained tubercle bacilli, and in six weeks a 5 cm. cavity developed.

Signs and Symptoms Accompanying Active Disease

Each person was carefully interviewed for a history of his disease in retrospect. In only one could the symptoms elicited be said to have been more than suggestive: an acute respiratory infection with a small hemoptysis.

There were two cases in which pneumonia was the presenting symptom. In each instance it was considered to be of the virus type early in its course.

Location and Spread of Lesions

In recent years, the importance and gravity of an infraclavicular lesion have been emphasized. Earlier it was held generally that in adults all pulmonary tuberculosis began in the apex, producing symptoms and signs of "consumption" as the lesion extended downward below the clavicle. In this small series, apparently inactive lesions in both apex and infraclavicular regions have been the starting point of active disease.

Years Between Employment and Tuberculous Activity

In mass surveys the question of frequency of reexamination arises. Do roentgenograms of the chest which are negative and "clinically not significant" for one, three or five years imply life-long freedom from active disease? The answer is indubitably no; repeated roentgenograms at regular intervals, after prolonged absences and in the presence of suggestive symptoms are indicated. The four persons in whom active pulmonary tuberculosis developed upward of 10 years after their employment had had minimal inactive infection throughout that period; the lesions had appeared hard, and serial roentgenograms had shown no change.

Duration of Treatment

It has long been known that the time required to arrest active pulmonary tuberculosis is closely correlated with the stage of disease when treatment is instituted. Six to 18 months is usually required to arrest minimal lesions. In only two cases of this series was this time exceeded.

All of the minimal infections were treated by rest alone. Pneumothorax was successful in controlling moderately advanced disease, and the person thus affected was returned to work after 18 months, therapeutic pneumothorax being maintained.

Tuberculosis Control in Industry, Charles R. Allison, M.D., Occupational Medicine, September, 1946.

PUBLIC RELATIONS

This is the first year that our Society has had a Public Relations Committee which is really functioning. It seems to have come on the scene as the natural result of a desire to combat certain elements and tendencies which have arisen, and it is a logical development by means of which we can sell the public on prepaid hospital or sickness insurance, or both; can put across the objectives of the North Carolina Medical Care Commission; and can build up good will generally. In the space of a few months this committee has become one of our most important agencies, and also one of our most expensive undertakings. The most potent means of acquainting the public with our views is through the press and radio, and both these mediums cost money. Yet not one of us would now feel that we could get along without this important committee. There is hardly an activity undertaken by our Society which can not be helped and supported by well directed public relations. Indeed, it would be difficult to put over many worthwhile activities without the spadework of the Public Relations Committee.

We must realize, as we never have before, that to maintain our position of influence and leadership, and to progress, we must ever be on the alert with well directed and appropriate publicity. No organization can stand aloof and not interest itself in public affairs or in politics if it is dependent on the public, as we are, for its very existence.

A case in point I would like to call to your attention: The last General Assembly appropriated funds which, with funds provided by the Hill-Burton bill, are to be used for the construction of a large teaching hospital at Chapel Hill and other necessary installations so that a four-year medical school can

be inaugurated. This act also made available funds which will supplement funds from the Hill-Burton bill to assist counties in the construction of health centers in rural areas. These provisions are all splendid and will go a long way toward providing medical care for the people of our state; but the Assembly made no provision for the maintenance of these agencies or for the salaries of professors and teachers in the new four-year medical school. It must be our duty, therefore, as physicians and as guardians of the health of our communities, to get in touch with our representatives and see that the next Assembly passes an enabling act to provide for a top-flight faculty and for the proper maintenance of the four-year medical school. The present act provides, as it should, that the counties in which health centers are to be located must provide funds to maintain the clinics or hospitals built. I ask you to keep up the good work this committee is doing. Strive to make the practice of medicine practical and the cost as reasonable as possible. Let us dispense with as many gadgets as we can without compromising our duty in determining the diagnosis. These gadgets are the things that make sickness expensive, and the more expensive sickness becomes the more certain people are to turn to government medicine as the only solution.

I will call your attention to a situation which has arisen in the past few years and which, if state medicine were thrust upon us, would certainly work hardships and injustice on physicians. The chief aim and purpose of a young physician just entering private practice or beginning his hospital training nowadays is to "get off his boards." During World War II, certification by a "board" seemed to be the important consideration in deciding the assignment and rank a medical officer should have. The alert young graduate, therefore, sees very clearly that in the event of another war or government control of medicine, the board diplomate would occupy the position of preference. Under state medicine, a baby delivered by the family doctor would probably cost from $35 to $50, depending upon the locality in which the patient lived; for a baby delivered by a board diplomate the fee would be $75 to $100. I know of one specialist who delivers babies only on Tuesdays and Thursdays. His fee would probably be $200.

Boards have their place, and have a wholesome influence in inducing young graduates to secure adequate hospital training before going into private practice. Experience gained in actual practice with a group or other specialists, natural ability, and the will to continue to learn and keep abreast of the forward march of medicine, however, are the criteria by which doctors should be judged in being evaluated for service to the public or for a government service or agency.

J. F. ROBERTSON, M.D.
Wilmington

BULLETIN BOARD

NEWS NOTES FROM THE DUKE UNIVERSITY SCHOOL OF MEDICINE

The Duke Eastern North Carolina Postgraduate Course will be held June 8-11 at the Duke University School of Medicine. Announcements will be sent to physicians in this area.

NEWS NOTES FROM THE BOWMAN GRAY SCHOOL OF MEDICINE OF WAKE FOREST COLLEGE

Dr. Henry Shepard Fuller, assistant professor of preventive medicine and attending physician of the VA regional office in Winston-Salem, has been awarded a fellowship in medical research by the Guggenheim Memorial Foundation. The project that he will pursue under the fellowship will involve studies of the taxonomy and distribution of mites of the family Trombiculidae in relation to their transmission of disease. During a six months' leave of absence from the medical school he will spend three months in Washington, D. C., and three months at the Rocky Mountain Laboratory of the U. S. Public Health Service at Hamilton, Montana.

* * * *

Dr. Lloyd J. Thompson, head of the department of neuropsychiatry, lectured in Richmond on April 16 at the final session of the Virginia Institute Seminar in Psychiatry and Neurology. His subject was "Organic Reactions." The Seminar was held under auspices of the Virginia Department of Mental Hygiene and Hospitals, the American Psychiatric Association, the Medical Society of Virginia, and the U. S. Public Health Service.

On April 23, Dr. Thompson and Dr. Richard Masland, also of the department of neuropsychiatry, went to the VA hospital at Fayetteville for lectures and clinics. Dr. Thompson's subject was "War Neuroses" and Dr. Masland's, "Intracranial Arterial Aneurysms."

* * * *

Dr. Harold D. Green, head of the department of physiology and pharmacology, attended sessions of the Scientific Council of the American Foundation for High Blood Pressure in Cleveland, Ohio, April 30 and May 1. Dr. Green is a member of the subcommittee which will outline a book to be published by the Foundation for those interested in supporting such research.

Dr. Frank Lock, head of the department of obstetrics and gynecology at Bowman Gray School of Medicine, was guest speaker for the section on obstetrics and gynecology of the Louisiana State Medical Society at the meeting in Monroe, La., April 12-19. His subject was "Recent Advances in Maternity Care."

* * * *

Dr. W. E. Cornatzer and Dr. Camillo Artom, of the department of biochemistry, presented a paper for the section on biochemistry and physiology at the forty-fifth annual meeting of the North Carolina Academy of Science, May 7 and 8 at Davidson College. Subject of the paper was "The Relation of Chemical Structure to the Stimulation of Lipide Phosphorylation."

* * * *

Dr. Robert B. Lawson, of the department of pediatrics, spoke on "Present Concepts Regarding the Spread and Treatment of Poliomyelitis" at the meeting of the Georgia State Medical Society in Atlanta on April 28.

* * * *

Dr. Wingate M. Johnson, professor of clinical medicine, was guest speaker at the eighty-first annual meeting of the State Medical Society of Texas in Houston, April 26 to 29. He spoke on "Pancreatitis" at the general meeting and on "The Training of Students as General Practitioners" at the clinical luncheon of the section on general practice. He also spoke on the "Relationship of the General Practitioner to the Overall Health Program" at a subsequent meeting of the section on general practice.

* * * *

Dr. MacDonald Fulton, professor of bacteriology, was re-elected secretary of the North Carolina Society of Bacteriologists at a meeting held at Duke University Hospital in Durham on May 1. Papers were presented at the meeting by Dr. Manson Meads, Miss Martha Chilton, Mrs. Thelma Tucker, and Dr. Fulton, all of the staff of the Bowman Gray School of Medicine.

News Notes from the State Board of Health

Dr. Carl V. Reynolds, State Health Officer, has been awarded life membership in the American Social Hygiene Association for outstanding services in fighting venereal diseases.

News Notes from the North Carolina Tuberculosis Association

Among those participating in the annual meeting of the North Carolina Tuberculosis Association, held in Raleigh on May 17 and 18, were Dr. Hillis L. Seay, president of the Association, and Drs. H. Stuart Willis, David T. Smith, B. B. Bagby, W. M. Peck, J. S. Hiatt, Josiah Trent, J. J. Combs, H. McLeod Riggins, W. Reece Berryhill, S. B. McPheeters, Carl C. Aven, A. Derwin Cooper, and Edward G. McGavran. Mrs. Paul P. McCain reported on the Christmas Seal Sale, which was the most successful in history. The total amount received from the sale was $367,414.54—10 per cent more than the 1946 total.

* * * *

Dr. Esmond R. Long, director of Medical Research and Therapy for the National Tuberculosis Association, has been named editor-in-chief of the **American Review of Tuberculosis**, to succeed the late Dr. Max Pinner.

Carteret County Medical Society

The Carteret County Medical Society held its regular monthly dinner meeting at the Morehead City hospital, the hospital being host. The scientific program consisted of a case report by Dr. Theodore Salter of Beaufort. Dr. J. W. Morris made a report for the committee from the society which appeared before the Board of County Commissioners on April 5, requesting more funds for the County Welfare Department to be used for hospitalization of indigent patients. According to Dr. Morris the request was favorably received.

Dr. Louis Hayman, of Cherry Point, was received as a new member of the hospital staff and the county medical society.

At the March meeting of the Carteret County Medical Society the matter of rebates and fee-splitting was strongly condemned, and the delegate to the State Medical Society was instructed to carry this sentiment before the House of Delegates in a very positive manner.

The officers of the Carteret County Medical Society are Dr. J. W. Morris, president, Morehead City, and Dr. Frank E. Hyde, secretary, Beaufort.

Reported by:
N. THOMAS ENNETT, M.D.
County Health Officer
Beaufort, N. C.

Catawba Valley Medical Society

A dinner meeting of the Catawba Valley Medical Society was held in Lincolnton on April 20. The program, which was arranged by the Lincoln County Society, consisted of three papers on the subject of gastro-intestinal hemorrhage by Dr. James F. Reinhardt, Dr. W. G. Page, and Dr. John H. Fitzgerald, and a case report by Dr. W. V. Costner.

Edgecombe-Nash Counties Medical Society

The April meeting of the Edgecombe-Nash Counties Medical Society, which was held in Rocky Mount on April 14, was devoted to an open discussion regarding practices of the Society.

At the suggestion of the Society, a blood bank and a cancer detection service have been set up in Rocky Mount. The former is sponsored by the Junior Chamber of Commerce and the American Red Cross, the latter by the Business and Professional Women's Club and the American Cancer Society.

Forsyth County Medical Society

The Forsyth County Medical Society held a dinner meeting in Winston-Salem on April 13. The program consisted of a symposium on cancer.

News Notes

Dr. Carroll C. Lupton has announced the opening of offices for the practice of general surgery in Greensboro.

National Gastroenterological Association

The thirteenth annual convention of the National Gastroenterological Association will be held June 7-10 at the Hotel Pennsylvania in New York. Members of the medical profession are cordially invited to attend.

National Conference of County Medical Society Officers

The Third National Conference of County Medical Society Officers will be held at the Palmer House, Chicago, on June 20.

The purpose of this Conference is "To Develop a Working Partnership Between the American Medical Association and Every Physician." Although the program is directed primarily to officers of the county medical societies, any member of the A.M.A. is welcome to attend and participate in the discussion.

Golf Tournament

The American Medical Golfing Association will hold its Thirty-second Tournament on Monday, June 21. The famous Olympia Fields has been reserved for the medical golfers' tournament. Dinner will be held in Olympia Fields Clubhouse, after which prizes will be awarded.

All male Fellows of the American Medical Association are cordially invited to become Fellows of the American Medical Golfing Association; write Secretary Bill Burns, 2020 Olds Tower, Lansing 8, Michigan for application blank.

University of Pennsylvania Medical Alumni

University of Pennsylvania Medical Alumni will hold a dinner at the Convention of the American Medical Association in Chicago, Wednesday, June 23, 1948, at the Lake Shore Club, 850 Lake Shore Drive. On arrival in Chicago, alumni should get in touch with Miss Frances R. Houston, executive secretary of the Medical Alumni Society, at the University of Pennsylvania registration booth.

Chicago Medical Society

The Chicago Medical Society is offering physicians of the country two postgraduate courses in September. A course in Hematology and Neurology will be given September 13-18, and another in Cardiovascular and Respiratory Diseases will be given September 20-25, 1948. The sessions will be held in Thorne Hall on Northwestern University Medical School campus. An outstanding group of teachers from all sections of the United States will make up the faculty.

Information may be secured by writing the Chairman, Committee on Postgraduate Medical Education, Chicago Medical Society, 30 North Michigan Avenue, Chicago 2, Illinois.

American Urological Association

The Southeastern Section of the American Urological Association announces receipt of a $1000 donation from Mr. and Mrs. William R. McEwen, Ft. Lauderdale, Florida. The fund is to be used to stimulate research on the problem of "Urinary Bladder Dysfunction." An award of $250 will be made for the best essay presented before the Annual Meeting of the Southeastern Section. Competition is open to men who have graduated from medical school within the past ten years. Further information may be obtained by writing to Dr. Russell B. Carson, 408 Sweet Building, Ft. Lauderdale, Florida, secretary-treasurer.

(BULLETIN BOARD CONTINUED ON PAGE 282)

Classified Advertisements

RESIDENT PHYSICIAN WANTED

Private hospital specializing in treatment of alcoholism and narcotic addiction desires high type person, male or female, for permanent position. Salary open.

Address C. B. T.,
P. O. Box 456,
Winston-Salem, N. C.

ASSOCIATE WANTED

WANTED: A young or middle-aged eye, ear, nose and throat specialist as an associate in a well established practice.

Address replies to Box 292, Wilson, N. C.

PATHOLOGIST AVAILABLE

Pathologist of long experience, certified in clinical pathology by the American Board of Pathology, available as director of hospital laboratories in North Carolina or Virginia.

Address "Clinical Pathologist"
Post Office Box 456
Winston-Salem, N. C.

BOOK REVIEWS

Treatment in General Practice. By Harry Beckman, M.D., Professor of Pharmacology, Marquette University School of Medicine, Milwaukee, Wisconsin. Ed. 6. 1129 pages. Price, $11.50. Philadelphia and London: W. B. Saunders Company, 1948.

Beckman's **Treatment** has made for itself a recognized place as a reference work on the latest and most approved methods of treating the conditions commonly seen in general practice. The sixth edition, just off the Saunders press, maintains the high standard set by its predecessors for reliability and readability. The author has kept the clear style and humorous touch which make reading the book pleasant as well as profitable.

The fact that the sixth edition contains some hundred pages more than the fourth and sixty more than the fifth gives some idea of the rapid advances being made in therapy. It is fitting that such an admirable work on treatment should be revised often enough to keep one reasonably well abreast with the remarkable progress of therapeutics.

A Primer of Cardiology. By George E. Burch, M.D., F.A.C.P., and Paul Resser, M.D. 272 pages. Price, $4.50. Philadelphia: Lea and Febiger, 1947.

This is one of the most desirable and useful of the many treatises on cardiovascular diseases. It is compact and yet reasonably complete in scientific detail. Especially good are the chapters on edema and cardiac failure.

The authors are to be complimented, and the book is to be highly recommended not only to students but to teachers and practitioners as well.

(BOOK REVIEWS CONTINUED ON PAGE 282)

American Academy of General Practice

APPLICATION FOR MEMBERSHIP

Name in full ..... M.D. Date .....

Office address ..... City ..... State .....

Residence address ..... City ..... State .....

Place and date of birth ..... Citizenship .....

Premedical education: College or university .....

Date of graduation ..... Degree .....

Medical education: Medical school .....

Date of graduation ..... Degree .....

Internship: Hospital ..... Date .....

....

....

Licensure: State ..... Date .....

....

Graduate training following internship:

Institution Date

Residencies or fellowships ....

....

....

....

Teaching appointments ....

....

Have you been engaged in the active general practice of medicine for 3 years or more (including military service) and are you now so engaged? .....

Membership on hospital staffs (past and present): (Include offices held and membership on committees)

....

....

....

Member of following county and state medical societies: .....

Membership in other medical societies: (Include offices held and membership on committees)

.....

.....

.....

References: (Three; preferably members of A. A. G. P.)

..... (Address) .....

..... (Address) .....

..... (Address) .....

(Over)

Educational activities in the past three years:

1. Scientific meetings attended: Approximate number of hours

County medical society meetings.....

.....

Hospital (educational meetings only)

.....

.....

Conventions—State Med. Soc., A.M.A., and Others (Specify—giving dates):

.....

.....

.....

2. Post graduate courses taken (Specify—giving dates):

.....

.....

.....

3. Military record in World War I and/or II:

4. Contributions to medical literature (Use separate sheet if necessary.)

I am applying for active membership and enclose herewith my check in the amount of $35.00 covering the $10.00 initiation fee and $15.00 for the first year's dues, and $10.00 for state dues.

I understand that the money will be refunded if my application is not accepted. In submitting this application I hereby agree to abide by the Constitution and By-Laws of the American Academy of General Practice and to accept the Board of Directors as the sole and only judge of my qualifications to be and remain a member.

I certify that I do not limit my practice to a single specialty of medicine or surgery.

.....M.D.
(Signature of Applicant)

(For Committee on Credentials)

Approved for active membership ☐

Appointment not recommended ☐

Appointment deferred ☐

Enclose check and mail to:
DR. ROSCOE McMILLAN, Secretary
Red Springs, North Carolina

AUXILIARY

TWENTY-FIFTH ANNUAL MEETING, WOMAN'S AUXILIARY TO THE AMERICAN MEDICAL ASSOCIATION

The twenty-fifth annual meeting of the Woman's Auxiliary to the American Medical Association will be held in Chicago, June 21-25, with headquarters at the Hotel LaSalle.

A most cordial invitation is extended to all women who are Auxiliary members or guests of physicians attending the convention of the American Medical Association to participate in all social functions and attend the general sessions. Whether Auxiliary members or not, the wives of doctors will be most welcome.

Auxiliary headquarters will be on the mezzanine floor of the Hotel LaSalle. All meetings and functions will be held at the Hotel LaSalle unless otherwise stated in the program, or announced during the meeting. Please register early and obtain your badge and program of the social function.

All tickets should be purchased soon after arrival. These will be sold at the Auxiliary headquarters. All meetings and social affairs will begin at the time scheduled. Please be prompt.

Registration Hours

Sunday	2:00 p.m. to 4:00 p.m.
Monday	9:00 a.m. to 4.00 p.m.
Tuesday	9:00 a.m. to 4:00 p.m.
Wednesday	9:00 a.m. to 4:00 p.m.

PROGRAM

Monday, June 21, 1948

4:00 p.m. Tea in honor of Mrs. Eustace A. Allen, President and Mrs. Luther H. Kice, President-Elect, Woman's Auxiliary to the American Medical Association, Century Room
Tickets $1.25. All doctors' wives cordially invited
Hostesses: Auxiliaries to the Illinois State Medical Society and to the Chicago Medical Society

Tuesday, June 22, 1948

9:00 a.m. Formal opening of the Twenty-fifth Annual Meeting of the Woman's Auxiliary to the American Medical Association, Illinois Room (mezzanine floor)

12:00 M. Luncheon in honor of the Past Presidents of the Woman's Auxiliary to the American Medical Association, Grand Ballroom (19th floor) Tickets $3.50
Guest Speaker: Morris Fishbein, M.D., Editor, *Journal of the American Medical Association* and *Hygeia.*

2:00 p.m. Afternoon session

8:00 p.m. Opening Meeting of the American Medical Association, Grand Ballroom, Hotel Stevens. Members of the Woman's Auxiliary and guests are welcome.

Wednesday, June 23, 1948

9:00 a.m. General Session of the Woman's Auxiliary to the American Medical Association, Illinois Room (mezzanine floor)

12:30 p.m. Annual Luncheon in honor of Mrs. Eustace A. Allen, President and Mrs. Luther H. Kice, President-Elect, Grand Ballroom (19th floor) Tickets $3.50

2:30 p.m. Afternoon session

Thursday, June 24, 1948

6:30 p.m. Annual Dinner of the Woman's Auxiliary for members, husbands and guests, Grand Ballroom (19th floor) Tickets $4.00

9:00 p.m. Reception and Ball in honor of the President of the American Medical Association—
Palmer House

Friday, June 25, 1948

Exhibits at Navy Pier

For Shy, Nervous, Retarded Children

Year round private home and school for girls and boys of any age on pleasant 150 acre farm near Charlottesville.

Individual training and care, expert teachers. Limited enrollment, amusements, special diets, medical care if necessary. Entrance made at any time. Write for Booklet.

Mrs. J. Bascom Thompson, Principal

THE THOMPSON HOMESTEAD SCHOOL

Free Union, Virginia

BOOK REVIEWS

(CONTINUED FROM PAGE 280)

Kompendium der Parasitischen Wuermer im Menschen. By Hans A. Kreis. 136 pages. Basel, Switzerland: Benno Schwabe & Co., 1947; imported by Grune & Stratton, Inc., New York.

Although the subject of parasitic helminths has been well covered in texts written in the English language, the present work is probably the most recent up-to-date book in German. A general section deals with parasitism; general morphology and biology of flukes, tapeworms and roundworms; the effects of these worms on the human host; general principles of therapy; and prophylaxis. Certain life histories are illustrated by diagrams. The second section of the book concerns diagnostic methods. General remarks on diagnosis are followed by considerations of serologic methods, complement fixation, precipitin and flocculation reactions, and intradermal tests. Examinations of stool, urine, sputum, blood, biopsy material, and the investigation of intermediate hosts are discussed. The author has presented useful keys for the identification of mature helminths, ova and larvae, accompanied by illustrations. Some of the illustrations of ova have been schematized, and are therefore possibly misleading.

In the third section of the book, the author treats six species of Trematoda (flukes), four species of Cestoda (tapeworms), and ten species of Nematoda (roundworms). Under each species he discusses geographic distribution, pathology, therapy, prophylaxis, and prognosis. This section is followed by a synopsis, listing the important synonyms of each species. Finally, certain important data are presented in tabular form, and the book is concluded by a short list of textbooks of helminthology.

The book can be recommended as a useful text, provided one has a knowledge of German and realizes that certain of the figures are schematic.

In Memoriam

WILSON PENDLETON, M.D.

Dr. Wilson Pendleton of Asheville, who died on Easter Sunday, was born at Portsmouth, Virginia, on February 22, 1886, and graduated in medicine at the University of Virginia in 1908. For some years he was on the staff of the Gaylord Farm Sanatorium at Wallingford, Connecticut. He was a captain in the Medical Corps during World War I and was stationed for two years at Camp Jackson, Columbia, South Carolina. From there he came to Asheville in 1919, where he was for several years associated with Drs. Minor and Ringer in tuberculosis work. For the last several years he devoted himself almost exclusively to allergy.

Dr. Pendleton was a member of the State Medical Society and of the American Medical Association; he was also a member of the American College of Chest Physicians and of the Southeastern Allergy Association. He was modest, shy, and self-effacing, one of the kindest of men, and very popular with his colleagues in Asheville.

"And thus he bore without abuse
The grand old name of 'gentleman';
Defamed by every charlatan
And soiled with all ignoble use."

Paul H. Ringer, M.D.

BULLETIN BOARD

(CONTINUED FROM PAGE 280)

AMERICAN HOSPITAL ASSOCIATION

Appointment of Charles T. Dolezal, M.D., superintendent of City Hospital of Cleveland, as assistant director and secretary of the Council on Professional Practice of the American Hospital Association, has been announced by George Bugbee, executive director. Dr. Dolezal assumed his new duties on May 1, filling the position left vacant with the resignation of Hugo V. Hullerman, M.D.

Dallas G. Sutton, M.D., Rear Admiral (MC) USN (Ret), director of study, Government Hospital Relations at the Washington Service Bureau, has been appointed in addition secretary of the Council on International Relations of the American Hospital Association.

PAN AMERICAN ASSOCIATION OF OPHTHALMOLOGY

Dr. Conrad Berens of New York was elected president of the Pan American Association of Ophthalmology at the Third Pan American Congress of Ophthalmology held in Havana, Cuba, January 4-10. It was decided to hold the next Congress in Mexico City in 1952.

The Congress adopted "statutes and bylaws," establishing the Pan American Association of Ophthalmology as a continuing organization with the objectives of fostering the progress of ophthalmology, establishing contacts among ophthalmologists of the Western Hemisphere, and promoting measures to conserve eyesight and prevent blindness among the peoples of the hemisphere.

ACADEMY-INTERNATIONAL OF MEDICINE

The Academy has for the first time compiled a comprehensive catalogue of surgical, medical and dental films, alphabetized both as to classification and titles, which will enable you to locate, select and secure pertinent films. It is limited in extent only by the number of films on which information was provided and contains a number of foreign films. In a section devoted to Audio-Visual Services are announcements of new developments and other data which might interest those who are building programs.

As a service to the profession the Academy is offering to mail, upon request, a complimentary catalogue to all A.M.A. officials, deans of medical schools, state secretaries, state program chairmen, authors of listed films and to certain foreign libraries. Copies will be provided to others, upon request, until the supply is exhausted.

Requests should be addressed to: Academy-International of Medicine, Department of Audio-Visual Aids, 214 West Sixth Street, Topeka, Kansas.

AMERICAN PHARMACEUTICAL MANUFACTURERS ASSOCIATION

Dr. Theodore G. Klumpp, president of Winthrop-Stearns Inc., was elected president of the American Pharmaceutical Manufacturers Association at its annual convention held in Havana, Cuba.